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AN ADEQUATE CALCIUM AND PHOSPHORUS DIETARY REGIME

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MUCH uncertainty and confusion regarding the mineral requirements of man and the indications for calcium and vitamin medication still exist. This is due, in part, to complexities inherent in determining what constitutes an adequate mineral intake under normal conditions and, more particularly, in disease; in part to conflicting and often exaggerated claims advanced for various pharmaceutical preparations. It would seem desirable, therefore, to consider the general problem of calcium and phosphorus requirements from two points of view: 1. What constitutes a diet adequate with respect to calcium and phosphorus? 2. If supplementary medication is thought to be indicated, how should the diet be so supplemented?

CURRENT ESTIMATES OF THE DAILY CALCIUM AND PHOSPHORUS NEED OF NORMAL HUMAN SUBJECTS

The maintenance requirement of normal human adults, calculated for 70 kg. body weight, was estimated by Sherman to average 0.45 gm. calcium per day.[1, 4] To this minimal need, Sherman suggests there be added a margin of safety at least as large as that generally allowed in the estimation of the protein requirement (50 per cent), raising the figure to 0.68 gm. calcium per day. Leitch[2] estimates the average maintenance requirement of calcium for normal adults at 0.55 gm. calcium per day; to which, it is stated, should be added, by way of "extra allowance," an amount presumably variable and, as yet, not determined.

In childhood, the calcium needs necessarily include both maintenance requirement and retention of calcium sufficient for normal skeletal growth. Sherman[1] suggests 1.0 gm. calcium daily as the average requirement for optimal

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calcium retention in growing children, an intake which ordinarily provides a mean daily storage of 0.01 gm. calcium per kg. body weight.[5] Estimates made by Leitch in this connection are given in Table I.

TABLE I

AVERAGE MINIMUM DAILY CALCIUM REQUIREMENT OF NORMAL CHILDREN AND ADULTS (AS ESTIMATED BY LEITCH[2])

AGE IN YEARS	GRAMS Ca DAILY
0.5–10	0.9–1.0
10 –15	1.4
15 –17	1.9
17 –19	1.1
19 –24	0.75
24 and over	0.55
Late pregnancy and lactation	1.45

The average daily phosphorus intake required for the maintenance of normal human adults is stated by Sherman[1] to be 0.88 gm. per 70 kg. body weight; for growing children to be 1.2 to 1.5 gm. phosphorus per child per day.

It should be emphasized that because of various uncertainties in the experiments which form their basis, and because of certain ambiguities in the definition of the term "requirement" as applied to nutritional needs, the figures cited above are approximations only. Moreover, they represent *mean* values for series of observations in which the range of individual variation was very large. For example, in the course of ninety-seven experiments which yielded an average daily value of 0.45 gm. calcium, Sherman encountered values as low as 0.27 and as high as 0.82 gm. calcium per day.[4] Bauer, Albright, and Aub[6] noted total calcium excretions varying in different periods from 24 to 65 mg. calcium per day in a normal subject maintained on a constant calcium intake of 135 mg. calcium per day; another normal subject of about the same weight was in calcium equilibrium during one period on an intake of 87 mg. calcium per day. It is probable that the variation in daily calcium requirement of the population at large is greater than that observed in this relatively small group of subjects. Certainly in pathologic conditions adversely affecting the absorption of calcium from the gastrointestinal tract, and in diseases associated with increased mobilization of calcium from the bones, the intake necessary for maintaining calcium balance greatly exceeds the average normal requirement.

ADEQUACY OF DIETARY SOURCES OF PHOSPHORUS

There appears to be a widespread belief that diets sufficient in calories and apparently well balanced—average mixed diets—can be assumed to be adequate with respect to calcium and phosphorus. And it is often taken for granted that if persons on such an "average" regimen develop certain disorders, particularly of the bones or teeth, then the diet must be supplemented by mineral and vitamin medication.

As regards the *phosphorus* requirement, recent studies in the field of nutrition do support the view that balanced diets sufficient in energy requirement are usually adequate with respect to phosphorus. Diets adequate in calories but not in phosphorus (such as are employed occasionally in the treatment of hypocalcemic tetany) often prove to be so unpalatable that patients reject them.

However, in some parts of the South where patent flour, degermed corn meal, sugar, and fats form the principal foods, such unbalanced dietaries, inadequate in phosphorus though sufficient in calories, are common.[3]

Phosphorus is widely distributed in foodstuffs—meat, eggs, wheat, legumes, milk, and nuts being particularly rich in this element (Table II)—and from all indications, the phosphorus needs of normal persons can be satisfied readily by proper dietary regulation. Recourse to supplementary phosphorus medication is rarely necessary; in fact, with the possible exception of low-phosphorus rickets and some types of osteomalacia, there appears to be no well-established specific indication for phosphate therapy. The common practice, in attempts to facilitate recalcification of bone, of supplying mineral or organic phosphates along with calcium salts has little experimental support and, in some instances, may lead to difficulties in absorption of the calcium administered.

TABLE II

MEAN CALCIUM AND PHOSPHORUS CONTENT OF COMMON FOODSTUFFS (FROM SHERMAN[1])

	PER CENT EDIBLE PORTION		PER 100 CALORIES FOOD			PER CENT EDIBLE PORTION		PER 100 CALORIES FOOD	
	Ca	P	Ca	P		Ca	P	Ca	P
Almonds	.252	.451	.038	.068	Hominy	.011	.070	.003	.020
Apples	.007	.012	.011	.019	Kale	.181	.067	.362	.134
Bananas	.008	.028	.008	.028	Lentils, dry	.102	.383		
Beans, lima, dried	.072	.386	.043	.112	Lettuce, headed	.017	.040	.091	.214
Beef, lean, 20% prot.	.013	.204			Liver	.011	.368		
Beets	.028	.042	.062	.093	Milk, cow's	.118	.093	.171	.135
Bread, white	.031	.097	.012	.037	Oatmeal	.065	.387	.016	.097
Butter	.016	.017	.002	.002	Onions	.041	.047	.084	.097
Cabbage, headed	.046	.034	.150	.090	Peanuts	.067	.395	.012	.071
Carrots	.045	.041	.097	.091	Pears, fresh	.023	.127	.022	.026
Cauliflower	.122	.060	.395	.194	Potatoes	.013	.053	.014	.057
Cheese, hard	.930	.701	.223	.168	Raisins	.060	.132	.017	.038
Cheese, cottage	.082	.263	.223	.168	Rice, white	.011	.099	.003	.029
Corn, sweet, fresh	.006	.103	.006	.096	Spinach	.078	.046		
Eggs	.063	.224	.042	.153	Tomatoes	.011	.029	.048	.127
Farina	.021	.125	.006	.035	Turnips	.056	.047	.164	.138
Figs, dried	.161	.116	.052	.037	Walnuts	.089	.358	.012	.050
Grapefruit	.021	.020	.048	.045	Watermelon	.007	.013		
Hazelnuts	.287	.354							

Average fish is estimated to contain per 100 gm. of protein, 0.109 gm. Ca, 1.1₇8 gm. P.
Average meat is estimated to contain per 100 gm. of protein, 0.058 gm. Ca, 1.078 gm. P.

ADEQUACY OF DIETARY SOURCES OF CALCIUM

As regards the *calcium* requirement, there is abundant evidence that apparently well-balanced diets, ample in energy requirement, may nevertheless be suboptimal, even grossly inadequate in calcium. According to Sherman,[1] "the ordinary mixed diet of Americans and Europeans, at least among dwellers in cities and towns, is probably more often deficient in calcium than in any other chemical element." A recent survey showed that about 16 per cent of typical American dietaries contained less than what appears to be the normal maintenance calcium requirement. Some of these diets were inadequate in total calories, but even when calculated on the basis of 3,000 calories daily, about 9 per cent of the dietaries would still be inadequate with respect to calcium.[1] Among growing children, and in selected localities, the incidence of dietaries deficient in calcium is doubtless higher. Of more than 4,000 unselected patients

whose food habits were analyzed at the New York Hospital by Bernheim,[7] in only two instances was the diet considered to be adequate with respect to calcium.

One may well inquire why, if these startling figures are to be taken literally, evidences of gross calcium deficiency (such as the "hunger osteomalacia" seen in the World War) are not more frequently observed in the population at large. Some infer that such evidences do exist but are not generally recognized. It is pointed out, for example, that growing children whose calcium intake is suboptimal are likely not to realize their full potentialities for growth and development, though they may not present abnormalities clinically. Marked stunting of growth in young rats maintained on diets adequate except with respect to calcium has been demonstrated by Sherman and his collaborators.[8, 9] It is pointed out further that in adults, the skeletal decalcification which occurs in old age so regularly[10] as to be regarded as a physiologic consequence of senescence may be, in reality, the result of moderate but prolonged deficiencies in calcium intake. Recent clinical studies indicate that advanced skeletal decalcification of this type—senile osteoporosis—occurs more commonly than was realized previously and may cause "rheumatic" pains, severe deformities, pathologic fractures and collapse of vertebrae with grave sequelae. Mineral deficiencies in diet doubtless play a role in the etiology of some cases of "senile osteoporosis," and as increased intake of calcium and vitamin D sometimes alleviates the condition, the importance of adequate dietary sources of these substances in prophylaxis seems evident. The long duration of time necessary for the development of clinically significant decalcification on average calcium-poor diets is emphasized by Leitch[2] whose calculations, which involve several problematic assumptions, to be sure, indicate a period of the order of ten years or more.

Whether or not these considerations adequately explain the discrepancy between the actual calcium intake of an appreciable proportion of the population and their (considerably higher) maintenance needs as estimated by metabolism experiments, is a matter for speculation. However, while a significant number of average dietaries may or may not meet *minimal* calcium needs, there is no doubt that many such seemingly adequate dietaries are *suboptimal* with respect to calcium. This is due to the irregular distribution of calcium in the various foodstuffs (Table II). Milk and milk products are rich in calcium which is present, moreover, in forms readily utilizable by the human organism; in "average" diets, it is estimated,[11] about two-thirds of the total calcium intake is derived from milk and cheese. Green leaf vegetables and nuts form an intermediate source of calcium whereas meats and fish are poor sources of this element.

The ingestion of sufficient amounts of milk and milk products affords the simplest and best way to insure adequacy in intake of calcium. A simple rule suggested by Sherman, and supported by virtually all subsequent study, states that for adults a pint of milk and for growing children a quart of milk incorporated in a balanced diet would satisfy the daily calcium requirements of normal persons.[1] Further, the increased calcium demands in pregnancy and lactation can be met in most instances by increasing the daily quota of milk and milk products.[12] There is a growing conviction that, in general, where prophylactic or therapeutic indications for increasing the calcium intake of normal

persons are thought to exist, this can be effected best not by the indiscriminate administration of pharmaceutical preparations, but by dietary regulation.

TABLE III

THREE REPRESENTATIVE DIETS PROVIDING MORE THAN ONE GRAM OF CALCIUM PER DAY (CALCIUM AND PHOSPHORUS CONTENT OF FOODSTUFFS CALCULATED AFTER SHERMAN[1])

DIET 1.	APPROXIMATE MEASURE	GRAMS	CHO	PROT.	FAT	CONTENT (IN %) OF Ca	P
Breakfast							
Sliced Orange	1 Medium	100	10	1	0	.024	.018
Toast	2 slices	60	32	5	1	.019	.058
Eggs	Two	100		13	11	.063	.224
Butter	3 teasp.	15			13	.002	.003
Hot chocolate	1 cup	200	16	6	9	.236	.186
Lunch							
Cr. aspar. soup	½ cup	100	8	3	8	.123	.106
Cheese	1 ounce	30	1	8	10	.279	.210
Head lettuce	Av. serv.	80	3	2	0	.014	.032
Carrots	½ cup	100	8	2	0	.045	.041
Bread	1½ slices	45	24	4	0	.014	.044
Butter	3 teasp.	15			13	.002	.003
Strawberries	⅔ cup	100	10	1	0	.034	.028
Cream	2 tablesp.	30	1	1	12	.027	.024
Dinner							
Roast beef	1 av. serv.	100		20	11	.012	.216
Baked potato	1 medium	140	22	3	0	.016	.064
String beans	½ cup	100	4	2	0	.055	.050
Bread	1 slice	30	16	3	0	.009	.029
Butter	5 teasp.	25			21	.004	.004
Baked custard	1 serving	150	10	10	10	.150	.205
	Total		165	84	119	1.128	1.545
			Calories 2067				
DIET 2.							
Breakfast							
Tomato juice	1 cup	200	8	4	0	.014	.030
Rolled oats	⅔ cup	20	15	2	1	.013	.077
Milk for cereal	½ cup	100	5	3	4	.118	.093
Eggs	Two	100		13	11	.063	.224
Toast	1 slice	30	16	3		.009	.029
Butter	2 teasp.	10			9	.002	.002
Milk	1 cup	200	10	6	8	.236	.186
Lunch							
Meat or fish	1 av. serv.	100		20	11	.012	.216
Potato	½ cup	100	19	2		.013	.053
Cabbage	½ cup	100	4	2		.046	.034
Parsnips	½ cup	100	18	2		.060	.076
Bread	1 slice	30	16	3		.009	.029
Butter	3 teasp.	15	0	0	13	.002	.003
Milk	1 cup	200	10	6	8	.236	.186
Chocolate blanc mange	½ cup	100	26	4	7	.102	.106
Dinner							
Poached egg	One	50		7	6	.032	.112
Spinach	½ cup	100	4	2		.078	.046
Cauliflower	½ cup	100	4	2		.122	.060
Milk	1 cup	200	10	6	8	.236	.186
Bread	1 slice	30	16	3		.009	.003
Butter	3 teasp.	15			13	.002	.003
Junket	½ cup	100	5	3	4	.118	.093
	Total		186	93	103	1.532	1.847
			Calories 2043				

TABLE III—CONT'D

DIET 3.	APPROXIMATE MEASURE	GRAMS	CHO	PROT.	FAT	CONTENT (IN %) OF Ca	P
Breakfast							
Stewed dried figs	Three	30	21	1		.048	.035
Rolled oats	⅔ cup cooked	20	15	2	1	.013	.077
Milk for cereal	½ cup	100	5	3	4	.118	.093
Egg	One	50		7	6	.032	.112
Toast	1 slice	30	16	3		.009	.029
Butter	2 teasp.	10			9	.022	.002
Hot chocolate	1 cup	200	16	6	9	.236	.186
10:00 A.M.							
Milk	1 cup	200	10	6	8	.236	.186
Lunch							
Omelet	Two eggs	100		13	11	.063	.224
Tomato	½ cup	100	4	2		.011	.029
Endives	1 av. serv.	80	3	2		.083	.031
Bread	1 slice	30	16	3		.009	.029
Butter	2 teasp.	10			9	.002	.002
Milk	1 cup	200	10	6	8	.236	.186
Sliced orange	1 medium	100	10	1		.024	.018
3:00 P.M.							
Cheese	1 ounce	30	1	8	10	.279	.210
Saltines	Three	10	7	1	1	.002	.010
Dinner							
Meat or fish	1 av. serv.	100		20	11	.012	.216
Mashed potato	½ cup	100	19	2		.013	.053
Cauliflower	½ cup	100	4	2		.122	.060
Bread	1 slice	30	16	3		.009	.029
Butter	4 teasp.	20			17	.003	.003
Rice custard	½ cup	100	13	7	7	.137	.169
Bedtime							
Milk	1 cup	200	10	6	8	.236	.186
Saltines	Three	10	7	1	1	.002	.010
	Total		203	105	120	1.957	2.185
			Calories	2312			

There are, of course, numerous exceptions to this general policy. Some persons have an unconquerable aversion to milk, a difficulty which can be circumvented, however, by selection of other foods rich in calcium (Table III, diet 1). There are people who are allergic to milk proteins and develop severe gastrointestinal or skin reactions to milk ingestion, a condition not rare in infants. Occasions arise, as in the management of some forms of hypocalcemic tetany, when it may be desirable to give calcium in forms other than milk because of its high phosphorus content. Under conditions adversely affecting the absorption of calcium from the gastrointestinal tract, in some diseases associated with increased mobilization of lime salts from the bones, it is often necessary to reenforce the diet with supplementary calcium and vitamin D medication. And in certain diseases characterized by absolute or relative calcium deficiency (hyperparathyroidism, for example) prevention or cure by increased dietary or medicinal administration of calcium is futile.

Two representative diets rich in calcium are included in Table III. Diet 2 affords an average intake of approximately 1.5 grams calcium, diet 3 an average of approximately 2.0 grams calcium per day. These estimates are based upon

the mean calcium content of the edible portion of the several foods included, according to representative analyses by Sherman[1] (Table II).

LIMITATIONS IN ESTIMATING THE CONTENT, AVAILABILITY AND UTILIZATION OF CALCIUM IN DIETARIES

In the application of these and similar dietaries, certain sources of variation and error should be recognized. The calcium contents of common foodstuffs indicated in Table II are averages of a number of determinations which show a wide scatter in the composition of many foods. This variation in the calcium content of individual food samples may result occasionally in a considerable discrepancy between the calculated calcium content of dietaries and their actual calcium content, as determined by direct analysis. The same dietary may show, upon periodic analysis, significant variations in calcium content from time to time. As there is little information now available regarding the frequency and degree of such divergencies between the calculated and observed calcium content of dietaries, we have summarized in Table IV our own experience in this regard.[13] These comparisons indicate that, apart from variations in calcium content of foods, the calculated estimates tend to exceed the determined values in the majority of diets, even when Sherman's revised tables[1] are employed.

TABLE IV

COMPARISON OF CALCULATED CALCIUM CONTENT OF DIETS (ESTIMATED AFTER SHERMAN[1]) WITH THE CALCIUM CONTENT FOUND BY DIRECT ANALYSIS

	NO. DIETS STUDIED	CALCULATED CONTENT > DETERMINED	CALCULATED CONTENT < DETERMINED	DISCREPANCY WITHIN ± 10% OF CALC. VALUE	DISCREPANCY > ±25% OF CALC. VALUE
High Ca content:	8	4	4	6	0
Intermediate Ca content:	4	4	0	1	0
Low Ca content	16	14	2	3	10

Even if the calcium content of foodstuffs is determined with precision, that proportion which can be assimilated by the normal human subject is variable and, for many foods, has not yet been investigated adequately. The degree to which ingested calcium is utilized by the organism depends upon many factors, one of the most important being the form of chemical combination in which calcium occurs in the various foodstuffs. In cow's milk, about one-third of the calcium content is in diffusible form, the remainder largely in combination with casein (and other milk proteins), and as calcium phosphate in an apparently colloidal state;[3] all of which permit virtually complete assimilation. The calcium content of some nuts (almonds) has been found to be in readily available form.[14] There are marked differences, however, in the assimilability of calcium derived from various vegetables and fruits: the calcium of carrots is largely utilizable[15] whereas that of spinach is, for the most part, nutritionally unavailable.[16] A variable proportion of the calcium content of plants is present as phytin, a calcium-magnesium salt of inositol phosphoric acid. Little is known about the availability of calcium from this source; the phosphorus content, however, has been shown to be poorly utilized.[17] In spinach, calcium occurs largely as the insoluble and physiologically inert salt of

oxalic acid. Fruits and vegetables also contain varying amounts of soluble calcium salts of weak organic acids (citric and malic) which are poorly dissociated but normally are converted to chloride, presumably, by the action of gastric juice.

Other extrinsic factors affect the availability of calcium in foods: the method of cooking with respect to leaching out of salts; the vitamin D ingested; the inorganic phosphate content and calcium-phosphorus ratio of the diet; the presence of foodstuffs affecting the pH of the intestinal contents, the pH of the food ash; the coarse fiber content of the diet; the relation in time of calcium-rich foods ingested to the remainder of the diet; and the like. The proportion of ingested calcium which is absorbed and retained by the organism (the "percentage utilization") depends further upon a number of intrinsic metabolic factors: the level of calcium intake and its relation to the calcium need of the growing or adult organism; the previous level of calcium intake; endogenous factors affecting bone metabolism; the pH of the intestinal contents as determined by intestinal secretions and bacterial flora; the gastric acidity; the digestion and absorption of fats. Present knowledge of many of these several factors is fragmentary, and the data in some particulars are conflicting or difficult to evaluate.

CONCLUSIONS REGARDING CALCIUM AND PHOSPHORUS NEEDS OF THE NORMAL HUMAN SUBJECT AND THE ADEQUACY OF DIETARY SOURCES OF THESE ELEMENTS

In view of the complexities indicated, it is evident that for the present, estimations of mineral requirements and nutrition can be only quasi-quantitative. The precise determination of minimal or optimal calcium or phosphorus needs in man is subject to limitations in method and definition, and to the limitation of marked individual variation. The calcium and phosphorus content of dietaries, as calculated from mean values assigned to the component foodstuffs, can be only approximate and leaves open the important questions of availability and utilization.

Nevertheless, certain generalizations seem justified by the nutritional data at hand. Normal phosphorus needs are met, ordinarily, by average mixed diets sufficient in calories. Many dietaries, however, even in persons free of economic restrictions or food idiosyncrasies, appear to be suboptimal with respect to calcium. This deficiency may be corrected by increasing the intake of milk and milk products so that for adults a pint of milk (or its equivalent) and for growing children a quart of milk per day is incorporated into a balanced diet. If such a regimen is followed, there would appear to be, at least for normal subjects, only rare need for supplementary calcium or phosphorus medication. The unnecessary substitution of pharmaceutical calcium and phosphorus preparations for foods, moreover, is not only costly but deprives the patient of other nutritional factors supplied by foodstuffs.

SUPPLEMENTING THE DIET WITH CALCIUM SALTS[18, 19, 20]

We are concerned here only with the oral administration of calcium salts and vitamin D when, as the result of deficiencies in alimentary absorption or in

parathyroid regulation, it is difficult or impossible otherwise to retain sufficient quantities from dietary sources. Hypoparathyroidism, rickets, osteomalacia, and sprue are well-recognized diseases in which this condition exists.

Of the various calcium salts suitable for oral administration, the lactate, gluconate, and phosphate are employed most widely, usually in milk. The lactate, containing approximately 13 per cent calcium, is soluble to about 10 per cent in cold water, is not unpalatable, is usually well tolerated and is of relatively low cost. The gluconate, containing approximately 9 per cent calcium, has the same advantages, but is more expensive. The phosphate is quite insoluble in neutral or alkaline solution, and it is doubtful that the provision of phosphorus "in the same proportion as in bone" affords more than a hypothetic advantage. Calcium chloride is a useful preparation, its slightly acidifying action being helpful under some circumstances, but it has a disagreeable taste, is upsetting to many patients and, because of deliquescence, does not keep well in bulk. A variety of complex organic salts of calcium are available, but no special advantage has been shown to accrue to their use, at least none in proportion to their added cost.

If the administration of calcium salts is to be effective, particularly in tetany or in attempts to recalcify bone, it is essential that large doses—10 to 20 grams a day—be given. In some patients, however, the protracted use of dosages large enough to be effective therapeutically may have undesired consequences, such as anorexia and gastrointestinal discomfort. Schour has called attention[21] to the occasional formation of scybala with obstipation and even intestinal obstruction as sequelae. Urinary excretion of increased amounts of calcium may predispose to the formation of concrements in the urinary tract. Because of these possible complications, it is evident that calcium medication in therapeutically adequate dosage should not be indiscriminate but should be restricted to those with well-defined indications. To prescribe calcium salts casually and haphazardly in tablets containing a few grains ("because it won't do any harm and might do some good") appears to be a not uncommon practice which is probably more futile and unnecessary than dangerous.

It is often desirable, further, to institute measures to facilitate absorption of calcium salts. Such measures are designed, for the most part, to decrease the pH of the intestinal contents in the small bowel, from the proximal part of which calcium salts are chiefly absorbed, and so to provide an optimal medium for maintaining the salts in solution. Dilute hydrochloric acid and acidifying salts such as ammonium chloride (in gram doses several times daily) are helpful for this purpose. Lactose in large doses (10 to 40 grams daily) in water or in buttermilk has proved beneficial, due probably to increased lactic acid fermentation in the bowel. The most effective adjuvant for increasing the absorption of calcium, however, is vitamin D.

SUPPLEMENTING THE DIET WITH VITAMIN D[22]

The chief sources of vitamin D in postnatal life are: 1. Absorption from the skin, following exposure of provitamin 7-dehydrocholesterol to short ultraviolet rays from the sun or from suitable lamps. 2. Absorption from ingested

foodstuffs or medicaments. Butterfat, whole milk, egg yolk, liver, and the flesh of certain oily fish supply the major part of vitamin D from ordinary dietary sources. Many average diets are poor in vitamin D and may prove deficient unless there is sufficient absorption from the skin or some supplementary source is ingested. The dietary of infants is particularly apt to be inadequate in vitamin D if not so re-enforced, and it is the general policy to incorporate some rich source of vitamin D into the infant dietary. Park[22] recommends that vitamin D be administered throughout the growing period, though it seems probable that the need of the child decreases with increasing age.

Under what circumstances the dietary of the adult should be supplemented with vitamin D, and how much should be given is still, however, largely a matter of surmise since the vitamin D requirement of the normal adult has not been established. It depends, apparently, not only upon the dietary intake but also upon the amount of exposure to short wave ultraviolet rays; upon the storage of vitamin D following such exposure; upon the adequacy of calcium and phosphorus intake; upon the secretion of bile into the gastrointestinal tract; and other factors. Park[22] is of the opinion that vitamin D medication should be recommended "for (1) all patients who by force of circumstances are deprived of the opportunity of obtaining the vitamin from the sun (night workers, factory workers, and persons confined indoors, in particular invalids), (2) for all persons whose diet is lacking in milk, the sole valuable source of calcium, or is generally poor, (3) for the aged, particularly when they are limited as to diet and confined indoors, (4) for persons suffering from fractures or having had operations on bones, and (5) for all pregnant women, no matter what the diet or the season." It is quite evident that here again a disproportion exists between the apparent need of vitamin D and the (seemingly smaller) amount actually available to a significant proportion of the apparently healthy population at large.

Of the preparations available for oral administration in the prophylaxis and treatment of vitamin D deficiency, cod liver oil is the most widely used. Many of the better brands are standardized at 200 U.S.P. units per gram, so that as much as 2,400 U.S.P. units per day may be provided by the administration of a teaspoonful (conveniently floated on orange juice) given three times a day; ample for the prevention of rickets in most infants and young children. Another convenient vehicle for the provision of prophylactic doses is vitamin D milk, of which there are three varieties: (1) Fortified, milk to which a vitamin D concentrate has been added directly, and standardized to contain 400 units per quart; (2) metabolized, milk from cows fed irradiated yeast, standardized to contain not less than 400 units per quart; (3) irradiated, milk exposed in thin films to ultraviolet light, and standardized to contain 135 units per quart. Many brands of evaporated milk are now irradiated, providing 135 units to the reconstituted quart. Vitamin D bread is also available, containing 460 units in the form of viosterol to the 24-ounce loaf.

For therapeutic purposes, as in the treatment of rickets and to raise the serum calcium above subnormal levels, it is desirable to use more potent vitamin D preparations. A number of cod liver oil concentrates are available in various forms, combined with viosterol, calcium compounds, and other vitamins, and

are standardized at convenient vitamin D levels. Percomorph oil, derived from the livers of fish species particularly rich in vitamin D, is widely used combined with cod liver oil in a preparation standardized to contain 8,500 U.S.P. units of vitamin D per gram. Viosterol (activated ergosterol) in oil and activated 7-dehydrocholesterol provide vitamin D in more concentrated form and in tasteless, hence easily administered, vehicles, but lack the high vitamin A content of many fish oils. A widely used preparation of irradiated, purified ergosterol in propylene glycol (miscible in water and given, usually, in milk) is standardized to contain 10,000 units of vitamin D per gram. A.T. 10 (dihydrotachysterol), another orally administered preparation[23] which promotes absorption of calcium from the gastrointestinal tract,[24] is employed particularly to raise the serum calcium in hypocalcemic tetany. It has little or no antirachitic action. Parathyroid extract is ineffective when taken by mouth and when given parenterally effects a rise in serum calcium chiefly or solely by mobilizing calcium from the skeleton rather than by facilitating the absorption of calcium from the gastrointestinal tract.

The use of very large concentrations of vitamin D and the use of dihydrotachysterol involve the hazards of toxicity and should be undertaken only when the dosage can be regulated under adequate control of serum calcium levels. The development of hypercalcemia is likely to be associated with anorexia, nausea, vomiting, lassitude, headache, frequency of urination and evidences of impairment of renal function. Because of the slow excretion of these sterols, such toxic manifestations following overdosage tend to persist several days to several weeks after discontinuance of medication.

SUPPLEMENTING THE DIET WITH MINERALS AND VITAMIN D IN THE ATTEMPT TO PREVENT OR TREAT DENTAL CARIES[21, 25, 26, 27, 28]

During the development of teeth in the prenatal and early postnatal life of man, metabolic disturbances (such as rickets), infections (such as syphilis) and toxic agents (such as fluorides) may result in hypoplasia of the enamel and related conditions. Whether or not hypoplasia of the enamel predisposes toward dental caries, whether or not systemic influences play a decisive role, or any role whatever in the development of dental caries, are still controversial matters. It is beyond the purpose and the capacity of the writer to attempt to sift the large body of evidence available on this subject, recently reviewed critically and in detail in several excellent studies.[21, 26, 27, 28] The prevailing view, based upon the consensus both of experimental and clinical evidence, is that local susceptibility is of primary importance in the etiology of dental caries, systemic factors, particularly in the adult, having at most only secondary or modifying effects; and that hypoplasia of the enamel does not predispose toward caries of the teeth.

This does not mean, of course, that in the prophylaxis and treatment of dental caries the general health and the calcium, phosphorus, and vitamin D requirements of the patient should be neglected. The carious subject, like the noncarious subject, should be provided with adequate sources of calcium, phosphorus, and vitamin D, particularly in childhood; and, as in the noncarious subject, this requirement is best fulfilled, in general, by proper foodstuffs according

to simple dietary rules. It is possible that, at least in childhood, adequate provision of nutritional requirements, particularly vitamin D, tends to lower the incidence of dental caries. Cowell[27] sums up the British experience in this regard by stating that "the resistance of teeth to decay can be raised by dietetic factors which affect their internal metabolism"—a reservation which limits the claim to few nutritional factors and to the period of development in which the internal metabolism of the teeth is susceptible to metabolic influences. Specific dietary restrictions of certain carbohydrate-rich foods[28*] may prove to have a more definite prophylactic effect on the development of dental caries. Once caries is present it is doubtful, according to the evidence at hand, that adequate or more than adequate intake of minerals or vitamins has any significant curative effect, at least in adults. It is generally agreed that the proper application of mouth hygiene and operative dentistry should not be delayed by such measures.

As to the use of calcium and phosphorus medication in the prevention and treatment of dental caries, the Council on Dental Therapeutics recently expressed the following opinion:[25] "There is no direct positive evidence from either the laboratory or the clinics that the addition of calcium or phosphorus compounds to an otherwise ample diet influences in any marked fashion the incidence of dental decay in the child or the adult; or that it promotes the development of noncarious teeth *in utero*, in infancy and during growth or in the adult; nor is there any sound evidence that a calcium drain during pregnancy and lactation is lessened by the addition of these compounds when the diet is well planned. On the contrary, there is evidence that such medication is unnecessary." A review of subsequent experimental and clinical experience relative to this subject failed to reveal any definite evidence to controvert this view.

SUMMARY

Current estimates of the calcium, phosphorus, and vitamin D requirements of the normal human subject are reviewed, with special reference to the adequacy of dietary sources. The general indications for supplementing the diet with medicational sources of calcium, phosphorus, and vitamin D, together with preferred methods of so doing, are considered.

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FACING FACTS OF FACE GROWTH

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I HAVE chosen the above title because I am going to do just what it says: face the facts of face growth. I shall outline not what we think we know, but what we do know, and how far it has taken us along the road to a clear interpretation of facial growth. In a very real sense I propose to take stock of our knowledge to date.

The first attempts made to interpret the nature of skull growth (including the face) assumed that it grew by simply swelling up like a balloon, with more or less equal radii in all directions. The "center" of skull growth was accepted as being somewhere near the earhole. Even in this rather naive interpretation, however, it was recognized that some of the radii grew faster, i.e., brain-case in the years of early childhood was obviously expanding more rapidly than face.

Then the anthropometrist—or, more properly speaking, the craniometrist—offered his aid. He picked out certain craniometric landmarks and used them as end points for a study of increase in dimensions, both absolute and relative. We come, then, to our first fact: techniques. We still must employ craniometry in our study of craniofacial growth.

Since the human face is three-dimensional, we are prepared for a threefold division into height, breadth, length (depth), each recording growth increments in its own plane, each accommodating the other. When we measure height we divide it into: 1) total, from nasion to gnathion; 2) upper, from nasion to prosthion; 3) dental, from prosthion to infradentale; 4) lower, from infradentale to gnathion. The sum of 2, 3, 4 is equal to 1. Upper face height includes the nasal and about three-fourths of the orbital height. Lower height is also mandibular height. When we measure breadth we divide it into: 1) bizygomatic, from zygion to zygion; 2) midfacial, from zygomaxillare to zygomaxillare; 3) palatal, from endomolare to endomolare; 4) bigonial, from gonion to gonion. The subtraction of 2 from 1 will give the amount of breadth contributed almost exclusively by the zygomatic arches; 2 includes nasal breadth. In measuring breadths the minimum frontal and biorbital and the interorbital must also be considered. In measuring lengths we accept the porion (or auriculare) as central and measure radii to nasion, orbitale, prosthion, infradentale, gnathion, gonion; or, if a perpendicular to the Frankfort horizontal is erected through the porion, we may measure the anterior or forward projection of these points. (Fig. 1.) To our length measurements we must add palatal length and, most important, the relatively intangible factor of forward drift of face due to cranial expansion at the junction of craniofacial sutures known as the "hafting zone."

From the laboratories of Anatomy and Physical Anthropology, University of Chicago.
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Truly this is a formidable battery of measurements, a long step forward from the simplicity of ballooning out. The face as a unit has been broken up into height, breadth, and length components; into orbital, nasal, dental, and alveolar areas; and fitted into a larger craniofacial adjustment complex. It not only grows in and of itself, but is also the recipient of contiguous growth impulse. Our techniques, still craniometric, are quite sound, for the basis of analysis is logical.

So much for techniques per se; how about their use? There are two methods, viz., the comparison of direct measurements and the calculation of percentage increment and the comparison of orthodiagraphic tracings. The former method is generally employed in the cross-sectional study: one group of 3-year-old children is averaged, a group of 4-year-old, and so on, and the trend is studied by the interpolation of different age series. The latter method finds use in the serial study. The tracing of a given age stage is superimposed on either an earlier or later, and size and proportional differences are noted.

Here, again, techniques plague us. There are no less than nineteen different methods of superposition, employing the resting position of the skull, base lines using craniometric landmarks, without and with the earhole as a center, and tracings of x-rays. The latter, developed by Broadbent, appears to be the most promising, possessing a variability less than that of purely craniometric points.

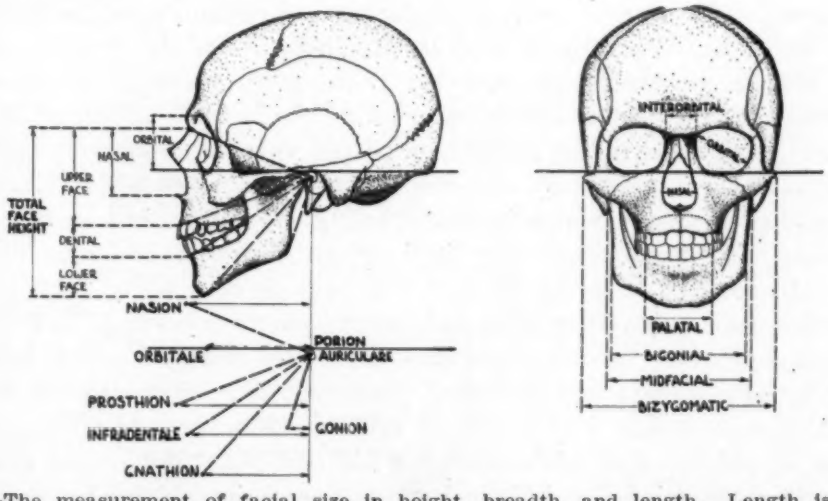

Fig. 1.—The measurement of facial size in height, breadth, and length. Length is shown measured radially from auriculare or projected from the vertical through the porion.

With the introduction of the x-ray we witness a merging of two major techniques: the purely craniometric, based on skulls alone; the purely cephalometric or gnathostatic, based on head and soft parts alone. Each has its limitations, but each surrenders its best to the x-ray. We are able, finally, to correlate the earlier craniometric and the later cephalometric into the all-inclusive roentgenographic. We still retain a logical basis of analysis, and we gain a wider scope of interpretation. It is possible to correlate craniometric and cephalometric studies not only by the x-ray, but by correcting

for soft tissue thicknesses. We may, for example, convert a craniometric orbitale into a cephalometric orbitale by adding 2.0 mm. for the thickness of the *M. orbicularis oculi.* In the same way we can locate the porion, or any other landmark, in both the skull and the head and correct the one to the other. This renders comparable the cranial studies of one author and the cephalic of another.

We may conclude, with a reasonable degree of confidence, that our techniques are factual. There still remains a residual problem: the time element. What units shall we set up to measure progress? Shall we turn to Hellman's tooth eruption age stages, Kronfeld's and Broadbent's tooth calcification criteria, Todd's skeletal maturational assessments, or shall we fall back on chronologic age? The first, admirable in pioneering studies, is too broad in scope; dental stages, e.g., from M2 to M3, mark relatively slight changes in the dentition while vigorous bony growth in the face is going on. The last disregards constitutional accelerations or retardations. Second and third are really two phases of the same general theme of development; both are a true register of bodily time, apart from sidereal time; both, in the hands of a skilled interpreter, will give a true estimate of achieved and present status. The ageing technique we may also accept as factual.

With this we proceed to the next fact: incremental growth in the three planes. Increase in total face height has periods of greater activity from birth to six months, three to four years, seven to eleven years, sixteen to nineteen years. The latter increment is largely respiratory and is sex-linked, greater in the male, associated with greater body bulk. In terms of decades height growth is slow in the first ten years, more vigorous in the second, lasting longer in the male. Increase in total face breadth is slow to the sixth year, increases, though less rapidly, until the years of puberty. Here, too, there is a midline respiratory element; there is also a postpubertal increase, more marked in males, which is almost solely zygomatic, i.e., an increase in the arches as a "buttressing" for the increased size of the muscles of mastication. Increase in total face length has periods of activity from birth to six months, four to seven years and sixteen to nineteen years. The first burst of activity provides for the deciduous arch; the second for M1, and, plus slower activity from seven to eleven years, for M2; the third for M3. In general, growth in face length is most vigorous from three to six years, with increase at puberty and adolescence for M2 and M3. The length growth is compound: actual increase in length, mostly posterior palatal for the permanent molars; forward thrust of facial skeleton in obedience to cranial expansion, probably localized in the sphenoidal complex. It is this forward drift that opens up the nasopharynx. There is also an adjustment of the relatively quiescent parts of the facial skeleton, probably brought about by molecular or interstitial change.

The foregoing paragraph has dealt solely with the upper face. The mandible, as far as we know, has its own pattern of growth, though some authorities feel that mandibular growth is a functional response to maxillary occlusal forces. Should mandibular growth be aberrant there may result an overshot

jaw, a receding chin, impacted third molars, or a permanent forward position of the condyle on the articular eminence.

We may now take stock of the fact of interplay or rhythm of growth in terms of major life periods: from birth to six years growth is vigorous in all directions; from six to twelve years growth is mostly in height, least in length; from twelve to twenty years there is an increase in length, and breadth exceeds height. In general length and breadth increase precedes changes in the dentition; height follows tooth eruption. The greatest percentage increase in height and breadth is between the eruption of M1 and M2. It is the interplay of growth in three planes that gives rise to the temporary, and entirely normal, imbalances that Broadbent calls the "ugly duckling" stages.

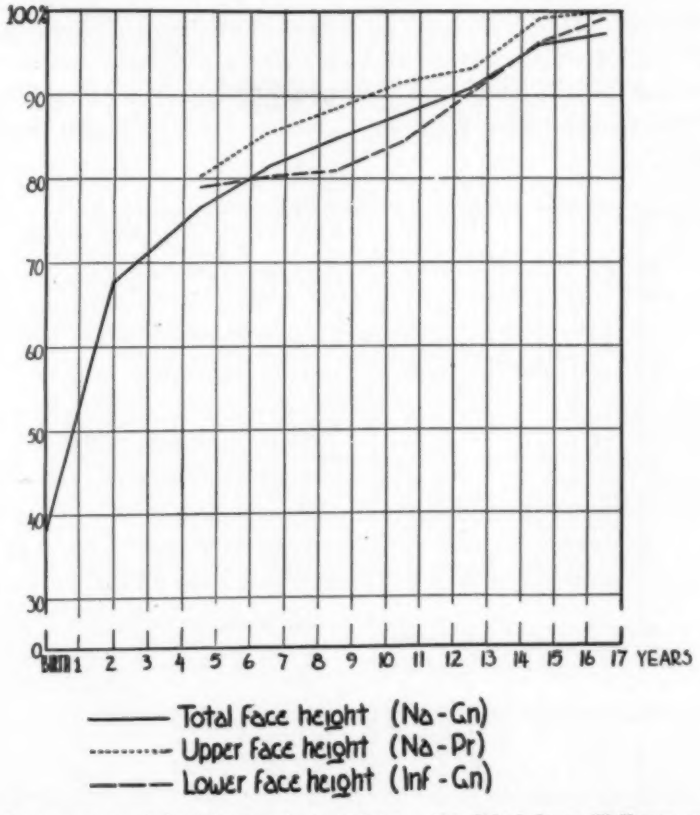

Fig. 2.—Percentage curves of facial growth in height. (Modified from Hellman and Goldstein.)

In the charts (Figs. 2-4) the fact of tremendous velocity of face growth is emphasized, as, is indeed, the essentially different nature of the three curves of height, breadth, and length. The important point is that at birth 40 per cent of adult height, 60 per cent of adult breadth, 70 per cent of adult length have been achieved. At two years the percentages are 70 per cent, 80 per cent, 75 per cent; at five years 80 per cent, 85 per cent, 85 per cent. From then on the remaining 15 to 20 per cent of growth is spread over 10 to 15 years. The avenues of growth are traversed rapidly until the blind alleys of growth cessation are reached.

The fact of attained growth does not preclude orthodontic treatment, for changes go on quite apart from growth, especially in the alveolar portion of the maxillary and mandibular arches. Part of the orthodontic response is an anteroposterior shift of the mandible, in a horizontal plane, due principally to a downward and backward rotation. Post-treatment accommodations consist in the readjustment of the occlusal plane and axial positions of the teeth. Orthodontics is esthetically successful in the child, clinically in the adult.

Incremental growth now must be looked at in the light of relative growth rates. The dimensions of the human face are greatest in width, less in height, least in length; the width increase is least, the length most. The length increase is greatest posteriorly; transverse increase is greatest inferiorly. Sex differences are found in the relatively longer female face, the relatively broader and deeper male face; the female jaw bones and dental arches are relatively more prognathous. If we consider craniofacial growth it is complete first in the head, then in face breadths, and last in height and length of the face. Variability of all facial dimensions increases absolutely with age, but decreases relatively. The variability is relatively greatest in face height, less in breadth, least in length.

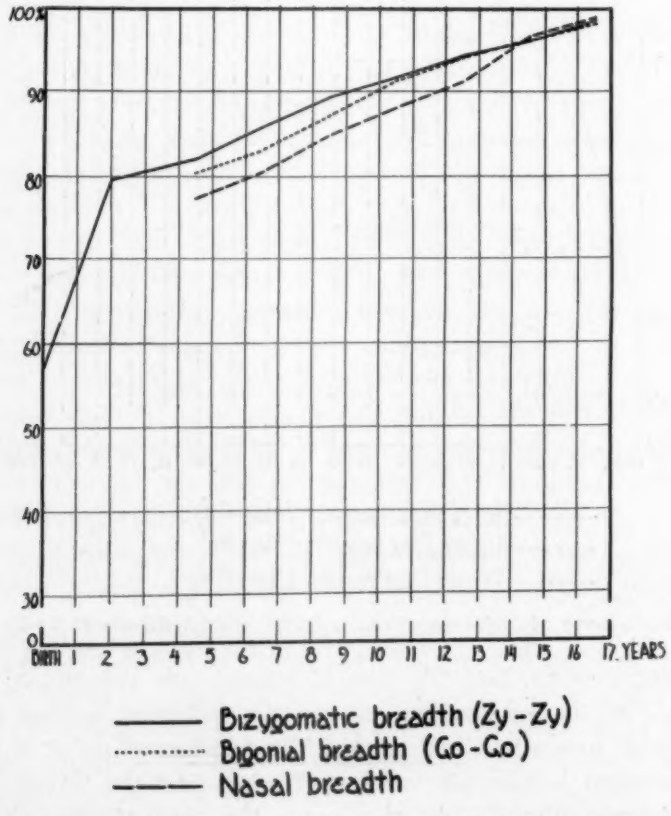

Fig. 3.—Percentage curves of facial growth in breadth. (Modified from Hellman and Goldstein.)

Normal growth is continuous but not uniform; there are spurts of activity. Furthermore, the growth of different parts in the same plane or the same part in different planes alternates in velocity and intensity. Finally, the rate of

growth is a rapid one. This combination of factors complicates interpretation but does not obscure basic fact. Our understanding of the rhythm of growth is fundamental to the assessment of a given stage of normal facial growth.

Now, the orthodontist is interested in normal growth for that represents his goal. What, he asks, about the fact of abnormal growth? We have only a few leads in this direction, based on actual studies of malocclusion. Upper face length is longer in all types of malocclusion except overbite; lower face is longer in retrusive bite and in Class II; midfacial length is shorter except in Class II; and lower facial length is shorter except in Class III. These rather meager bits of information give us one major conclusion: that malocclusion is a fundamental imbalance between forward or length growth of upper or midface and lower or mandibular face. Obviously, since here growth is such a factor, there must be a distinct correlation in children between success of orthodontic treatment and the uniform progress of growth.

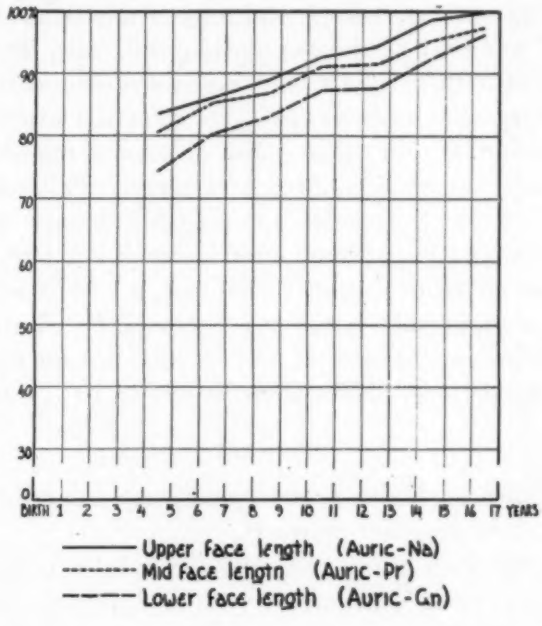

Fig. 4.—Percentage curves of facial growth in length (radial from auriculare). (Modified from Hellman and Goldstein.)

With this we come to the problem of etiology, and we are at once on relatively unstable ground. If we accept the principle that facial growth is subject to the same general influences and interferences as general bodily growth, then whatever inhibits the latter is reflected in the former. This rule does not hold absolutely, for although forward growth of the face is dependent on bodily development, vertical growth is more closely related to bodily size, a phenomenon more independent of the process of maturation. We may conclude, however, that face growth is expedited, retarded, warped, or inhibited by general constitutional ill-health or endocrine imbalance. The effect may be secondary rather than primary, as, for example, when allergy may temporarily enlarge adenoids and turbinates, leading to mouth habits productive

of malocclusion. Further, the constitutional or endocrine disorders may cause a halisteresis or demineralization which will, in the alveolar area, defeat attempts at local adjustment.

Despite all interruptions, however, growth does tend to a structurally or functionally adequate whole. The increased growth velocity in the postillness periods repairs defective facial growth, though in severe cases it does not obliterate its existence. It is this tendency to perfection and repair that holds hope for orthodontics.

In the foregoing paragraphs reference has been made to the normal, however we may define it: esthetically, etiologically, anatomically, functionally, or biometrically. Actually we may have recourse to still another concept, i.e., the so-called individual normal, wherein each child has a growth pattern (to be determined by serial study) uniquely its own. In practice, however, we have recourse to statistics to give us an idea, not of an absolute, but a relative normal. Variation is the rule, and its bounds may be assessed. Therefore, we accept as normal an average and plus or minus its standard deviation, between which 67 per cent of the population will fall. This large midgroup constitutes the great bulk in whom increase in size and change in proportions result in a symmetry that does not call for remedial attention. The 33 per cent that fall outside of the mean, plus or minus one standard deviation ($M \pm \sigma$), are the subjects of corrective treatment. This may be graphically illustrated as follows: at 3 years of age, in white males, the distance porion to nasion is 80.6 mm., with a standard deviation of 4.1 mm. This means that the range of one standard deviation (or sigma) is 76.5 to 84.7. All 3-year-old white males who fall in this range are "normal." A white child with a nasioporion dimension, at 3 years, of 70.0 or 90.0 has an abnormally short or abnormally long upper face. Here there is apt to be a disharmony.

SUMMARY

1. The techniques of facial growth study are soundly based on craniometric, x-ray, and maturational methods.

2. Growth in the face is in three planes: height, breadth, length.

3. Incremental growth is rhythmic, with an interplay between the several components, each with its own rate of growth.

4. Differential length growth in upper and lower face leads to malocclusion.

5. Face growth is susceptible to the same growth impulses or retardations as is body growth.

6. The concept of the normal is best understood in terms of a predictable statistical variability.

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TEMPOROMANDIBULAR JOINT DISORDERS

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IT IS conceded by clinicians and students of temporomandibular joint disorders that the roentgen ray is indispensable in the diagnosis of joint disorders and their complications. When properly carried out, it is the most accurate means of determining the condition of the joint.

Pains may be centered in the region of the joint in the absence of any roentgen ray findings. Pains having origin in other regions may be referred to the external and middle ear because of its extensive nerve supply from the fifth, ninth, and tenth cranial nerves, as well as from the second and third cervical. Recognition of this fact must be given consideration in making a differential diagnosis.

The following suggestive symptoms encountered are presented to show the importance of a differential diagnosis, eliminating factors that are not temporomandibular in origin.

It has been observed that unerupted mandibular third molars frequently are a source of earache, and more infrequently the maxillary third molars. A chronic alveolar abscess of the mandibular teeth may produce an earache on the same side. In tic douloureux, in which the mandibular division of the trigeminal nerve is affected, the pain may be centered in and around the ear. In glossopharyngeal neuralgia, rarely seen, but when present, pains radiate from the throat to the ear.

Infection arising from root tips involving the mucous membrane of the maxillary sinus may cause pain to be referred to the temporomandibular joint.

Exostosis of the maxilla has been observed as causing referred pains and discomfort when a denture has been placed over enlarged tuberosities of the maxilla, restricting normal limitations of movement and changing the condyle movements from normal excursions to a hinge movement.

Diagnostic symptoms of temporomandibular joint disturbances may be characterized by pains and tenderness in the joint and around the region of the ear; a snapping noise while chewing; tiredness in the region of the joint after meals; symptoms of a burning sensation on the side of the tongue; a change in the flow of saliva, either an increased or decreased amount; certain degrees of dizziness, often accompanied by headache, radiating posterior to the ear, and stuffiness or low buzzing sounds in the ears.

Seaver[1] has stated that the diagnosis of temporomandibular joint disorders is simple, but the difficulty is in being able to evaluate their importance as a factor in ear disturbances.

Joint disorders are less frequently associated with normal occlusion. Disturbances of the joint may begin to appear as the posterior teeth are lost in the

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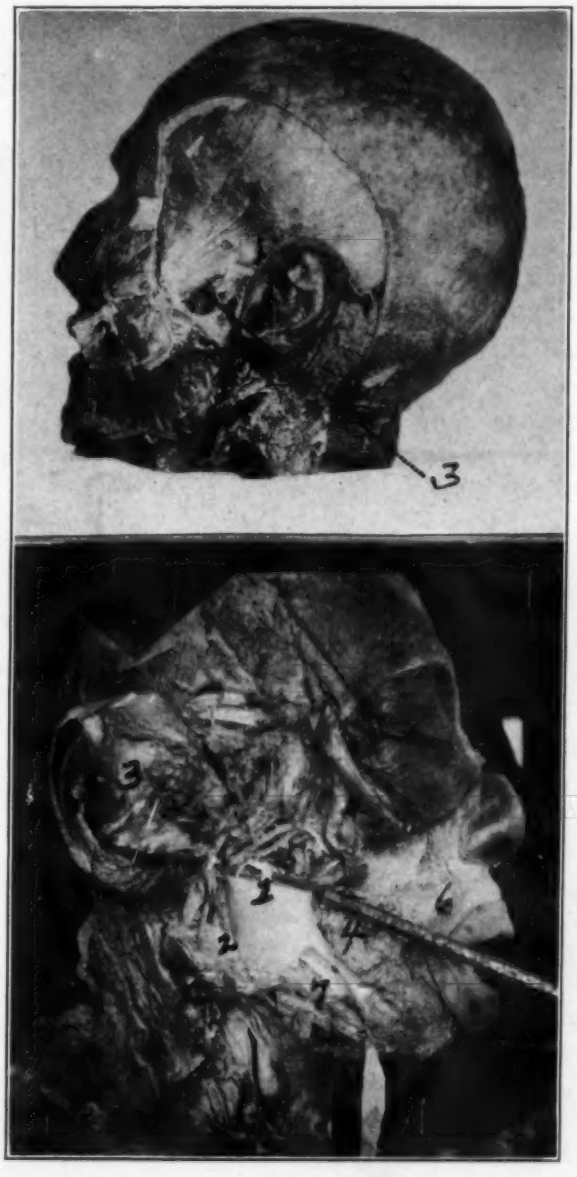
dental arches, either unilaterally or bilaterally, resulting in an overclosure or unbalancing of the condyle within the glenoid fossa, producing pain.

Overclosure has been observed to cause an impairment of hearing. The mere fact that hearing does improve with the repositioning of the condyles would merit some consideration of the anatomic structures which may be involved. It would be reasonable to expect that nerves capable of transmitting stimuli, which are in close range to the moving condyle within and immediately around the glenoid fossa, would be affected first in overclosure.

A.



B.

Fig. 1.—*A*, Deep dissection of temporomandibular joint. 1. Posterior deep fibers of temporal muscle; 2. zygomatic arch with origin of masseter muscle reflected anteriorly; 3. Insertion of external pterygoid muscle into fovea of condyle and into meniscus. *B*, Center of axis of joint is midramus for mandible under consideration. 1. Ramus was removed 1 mm. above lingula, insertion of sphenomandibular ligament; 2. stylomandibular ligament insertion; 3. zygoma reflected posteriorly; 4. buccinator muscle; 5. internal pterygoid muscle; 6. orbicularis oris muscle; and 7. mandibular nerve.

Since the method of approach to this problem has been first from the anatomic (Fig. 1, *A* and *B*) and second from the roentgen ray in clinical cases (Fig. 2), it will be necessary to give some consideration to the anatomy of the region involved.

The glenoid fossa is divided into two parts by a small slit called the petrotympanic fissure, extending mesially and laterally, being nearly parallel with the head of the condyle when the teeth are in normal occlusion, but at a depth of 2.5 cm. the condyle head is more parallel than at a lesser depth.

In making a detailed anatomic study of the temporomandibular joint, having in mind those factors that may cause ear disturbances and referred pains, attention is immediately attracted to nerves which might be irritated by pressure of the moving condyle.

Fig. 2.—Laminagram: Left lateral view of a clinical case taken at a depth of 2.9 cm., showing the petrotympanic fissure. (Courtesy of Sherwood Moore, M.D., The Edward Mallinckrodt Institute of Radiology, Washington University, St. Louis, Mo.)

The chorda tympani nerve, accompanied with the anterior tympanic artery, passes through the fissure, the artery supplying the anterior two-thirds of the tympanic membrane, the nerve passing between the malleus and incus to join the seventh or facial, and connecting it with the lingual beneath the external pterygoid head.

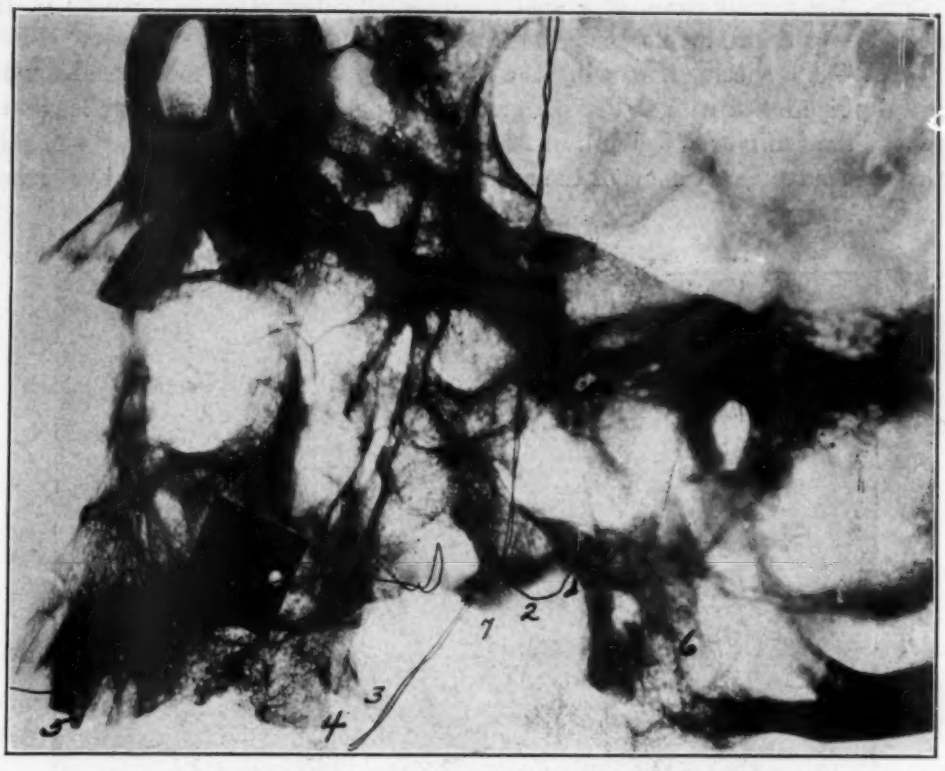


Fig. 3.—Radiogram of a specimen, showing the relative position of the chorda tympani as it enters the petrotympanic fissure (1); 2. condyle of mandible removed from glenoid fossa; 3. lateral pterygoid lamina; 4. tuberosity of maxilla; 5. canine root fragment; 6. mastoid cells; and 7. articular eminentia.

Fig. 4.—Prepared section demonstrating sagittal plane taken with the laminagraph, cut at 2.5 cm. from the zygomatic arch. The cephalic index is of assistance in predetermining the level to be used for each clinical case. 1. External pterygoid on fovea of neck of condyle; 2. auriculotemporal nerve passing mesially to the neck of the condyle; 3. internal pterygoid origin; 4. eminentia articularis; and 5. insertion into articular disk. (From Bielker: J. A. D. A. September, 1938.)

Fig. 3 is a radiogram of a specimen showing the relative position of the chorda tympani and its closeness to the moving condyle.

Fig. 4 is a specimen showing the relation of the condyle to the ear, also the sagittal plane taken with the laminagraph. The chorda tympani nerve leaves the range of the condyle at a depth of 2.5 cm. to enter the temporal bone to pass between the malleus and incus of the middle ear before joining the facial or seventh cranial nerve.

Fig. 5.

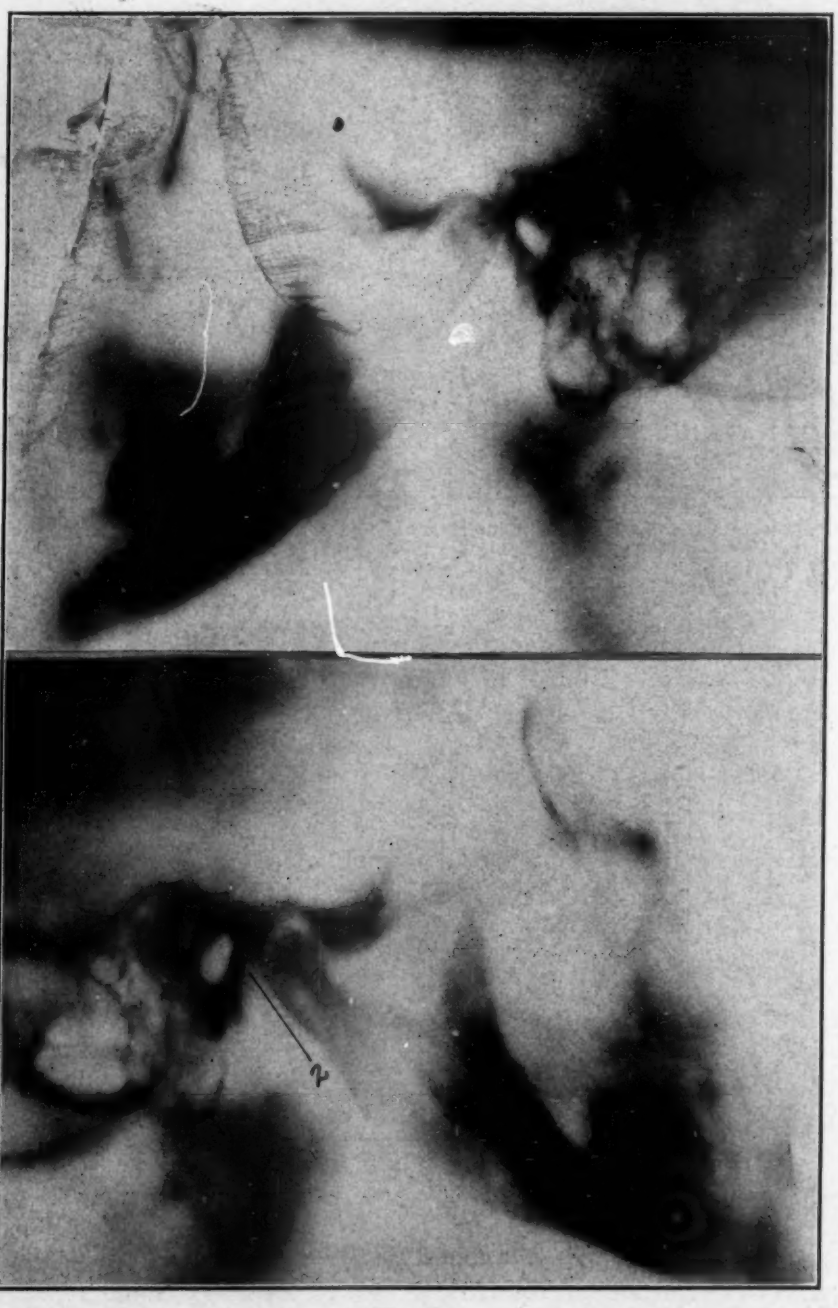


Fig. 6.

Figs. 5 and 6.—Laminagram of a clinical case. Above, left lateral taken at 2.6 cm.; below, right lateral taken at 2.6 cm. Observe the closer relation of the left condyle to the fissure (1) than that of the right side (2).

In those cases of overclosure in which the head of the condyle approximates the posterior wall of the glenoid fossa, the chorda tympani could easily be irritated, and be a factor in producing an ear disturbance.

Fig. 5 is a laminagram of a clinical case showing an overclosure after wearing a denture for two years. Note the close relation of the condyle to the fissure. Because of this, some significance should be placed upon the petrotympanic fissure. Since the chorda tympani nerve contains both motor and sensory fibers, a slight irritation at the point of its entrance into the fissure by the condyle could stimulate these fibers and produce a disturbance in the middle ear. Whether this disturbance is produced directly through the sensory fibers or through reflexes would not change the condition. The point of stimulation would remain the same, occurring on the mesial side of the glenoid fossa.

Fig. 7A.—Laminagram, clinical case, right lateral view.

Since sensory fibers within blood vessel walls have been demonstrated, it is reasonable to suppose that these fibers are contained within the anterior tympanic artery, which supplies the greater portion of the tympanic membrane. Irritation of these fibers alone might be sufficient to produce ear disturbances. In extreme abnormal cases, in which the cartilage has been partially destroyed permitting the head of the condyle to ride upon the glenoid wall, a change in blood pressure of the artery leading to the membrane might be a contributing factor.

This clinical case (Fig. 6) has been previously reported. However, it is the first case of the protrusive type of mandible in which the laminagraph was used, so it is being presented to demonstrate the principles involved in overclosure.

The patient was a male, 43 years of age, upon whom a maxillary alveolectomy had been performed two years previously. Dentures had been constructed. The resorption during this period had produced an overclosure, and the vertical dimension was lost. The patient complained of tiredness and slight pains in the region of the joint; occasional snapping noises were heard, and a sensation of stuffiness in the ear.

This case showed the necessity of the conservation of the maxilla in preparing the maxillary process for dentures. The unusual obtuse angle formed between the ramus and the body of the mandible was to be observed for a patient of this age. The meniscus apparently was very thin. In placing bite blocks in the various positions to bring the condyle heads forward and downward, it was demonstrated that the mere opening of the bite did not accomplish the desired result and that other principles were involved.

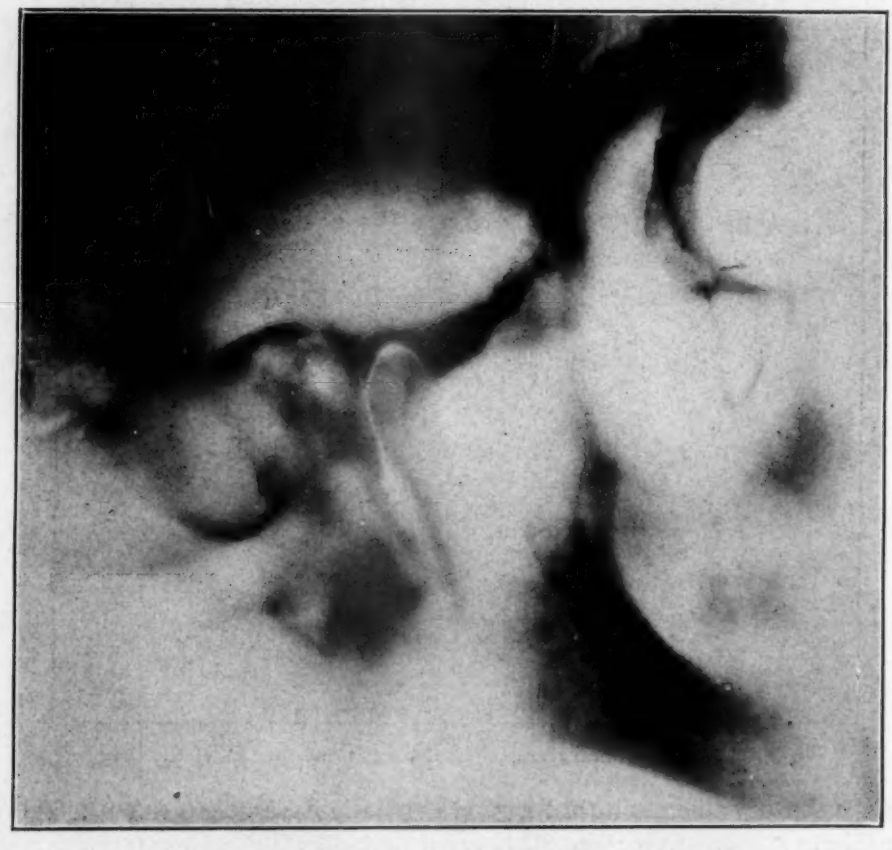


Fig. 7*B*.—Laminagram, clinical case, left lateral view.

The occasional snapping noise heard was due to extreme thinness of the meniscus. The looseness of the joint was attributed to overclosure caused by the early loss of posterior teeth on one side, and accounted for the occasional stuffiness in the ear.

The change in depth as indicated by the laminagraph on the lateral view of the head of the condyle indicated there was a wide and shifting position in the closure of the mandible. While this was not observed clinically in this case, it

would be concluded, inasmuch as the depth taken was 2.5 cm. and was constant in the series of records. The gradual overclosure which had developed in the past two years was evidently a result of removing the thin septae of the maxillary process, and placing the head of the condyle in close relation to the fissure.

Figs. 7 *A* and *B* is a completed case of a man aged 65, whose posterior teeth had been missing on the left side for several years. There was poor occlusion on the opposite posterior teeth; the remaining teeth showed signs of abrasion. Slight pains were present in the vertex region; there was an increase in the flow of saliva, and low sounds were not audible in the left ear. The correct relationship was established between the mandible and the maxilla, placing the condyles anterior to the petrotympanic fissure; the occlusion was balanced, and the dentures were completed. The patient reported improvement in hearing after wearing the dentures for six weeks.

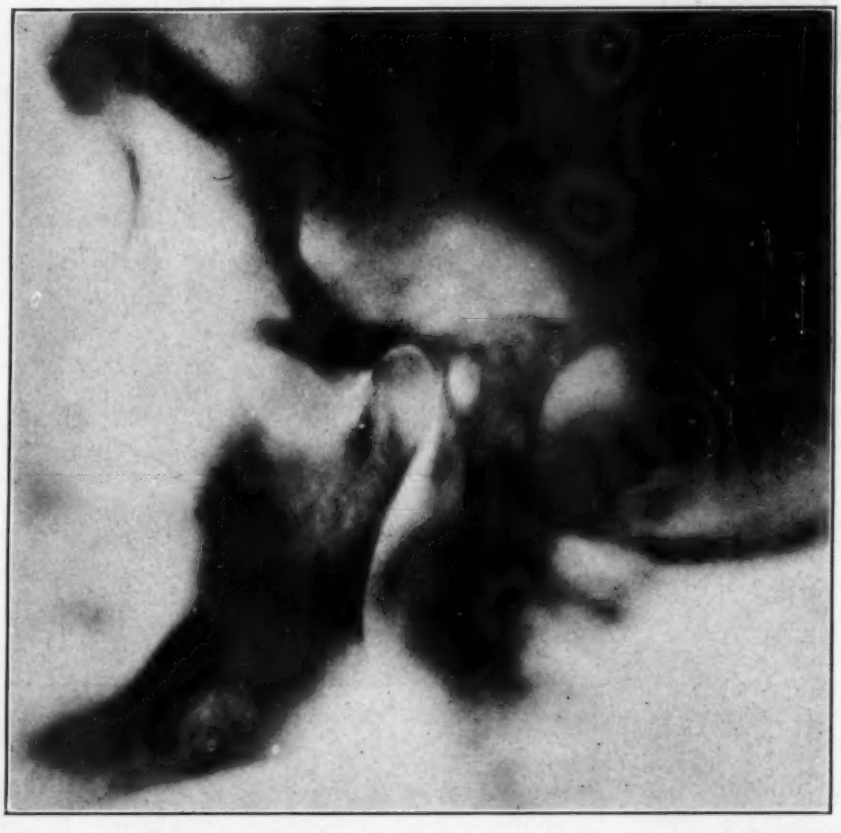


Fig. 8*A*.—Laminagram of the same clinical case as seen in Fig. 7*A*, ten months later, right lateral view.

Figs. 8 *A* and *B* is a check-up of the case ten months later which shows definitely the heads of the condyles maintained the same relationship to the fissure. The dentures were functioning successfully. Patient reported slight additional improvement in hearing. There was no further salivary disturbance.

In this pathologic joint there was evidence of irritation of the chorda tympani by the head of the condyle in making its mesial movement within the

glenoid fossa. This apparently accounts for the low hum reported and the activation of the salivary glands.

Valuable information may be obtained from either radiographic or laminagraphic records relative to the joint, which may be an aid in diagnosis. However, these will not have much value in making corrections unless we are able to determine the exact position of the condyles before beginning treatment, and to observe the changes made as the treatment progresses, in other words, to reproduce an exact record at any time and to see the effect the change in occlusion has upon the articulation.

Scientific records should be made of the temporomandibular joint before any radical changes in the occlusion are attempted, especially in cases requiring extensive reconstruction.

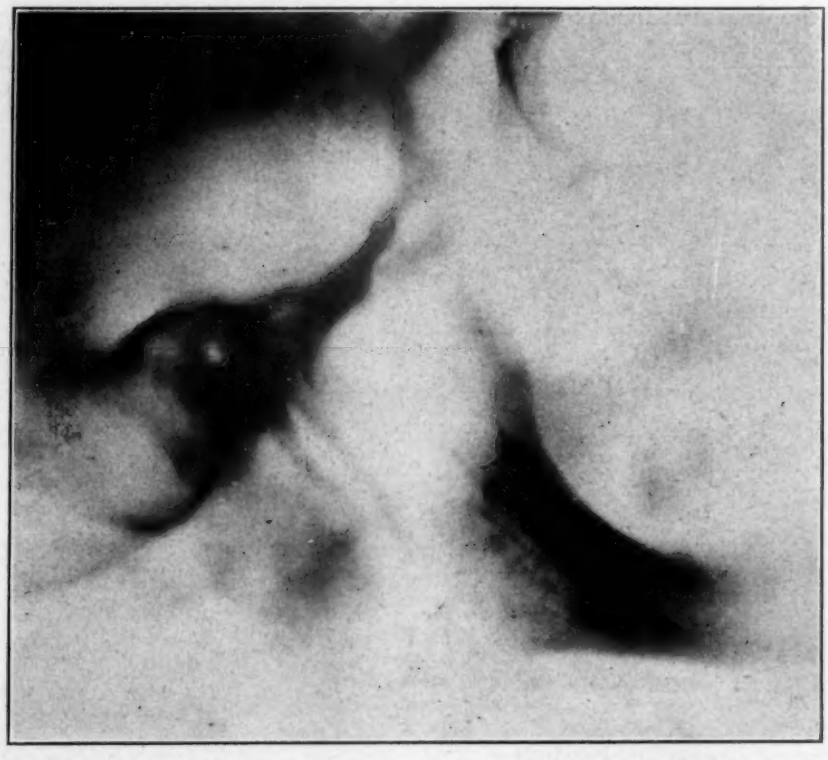


Fig. 8*B*.—Laminagram of the same clinical case as seen in Fig. 7*B*, ten months later, left lateral view.

The difficulty some operators have experienced is that in making the changes in the occlusion, and not being able to observe any changes in the joint, they have discredited the importance of using radiograms.

To be of value, these records must not only show comparative changes, but exact changes, at the beginning of treatment, during the course of treatment, as well as the final results obtained. With this thought in mind a clinical case is presented.

Fig. 9 is a clinical case of a man, aged 41 years. This case is presented to show first the effect of resorption of the maxillary process due to pyorrhea, causing a changed relationship between the mandible and maxilla, the beginning of

an overclosure, and second, its effect upon the changed relationship of the head of the condyle to the petrotympanic fissure in the glenoid fossa.

The patient complained of headaches radiating toward the region of the eye. All the teeth were radiographed and showed destruction of bone. The

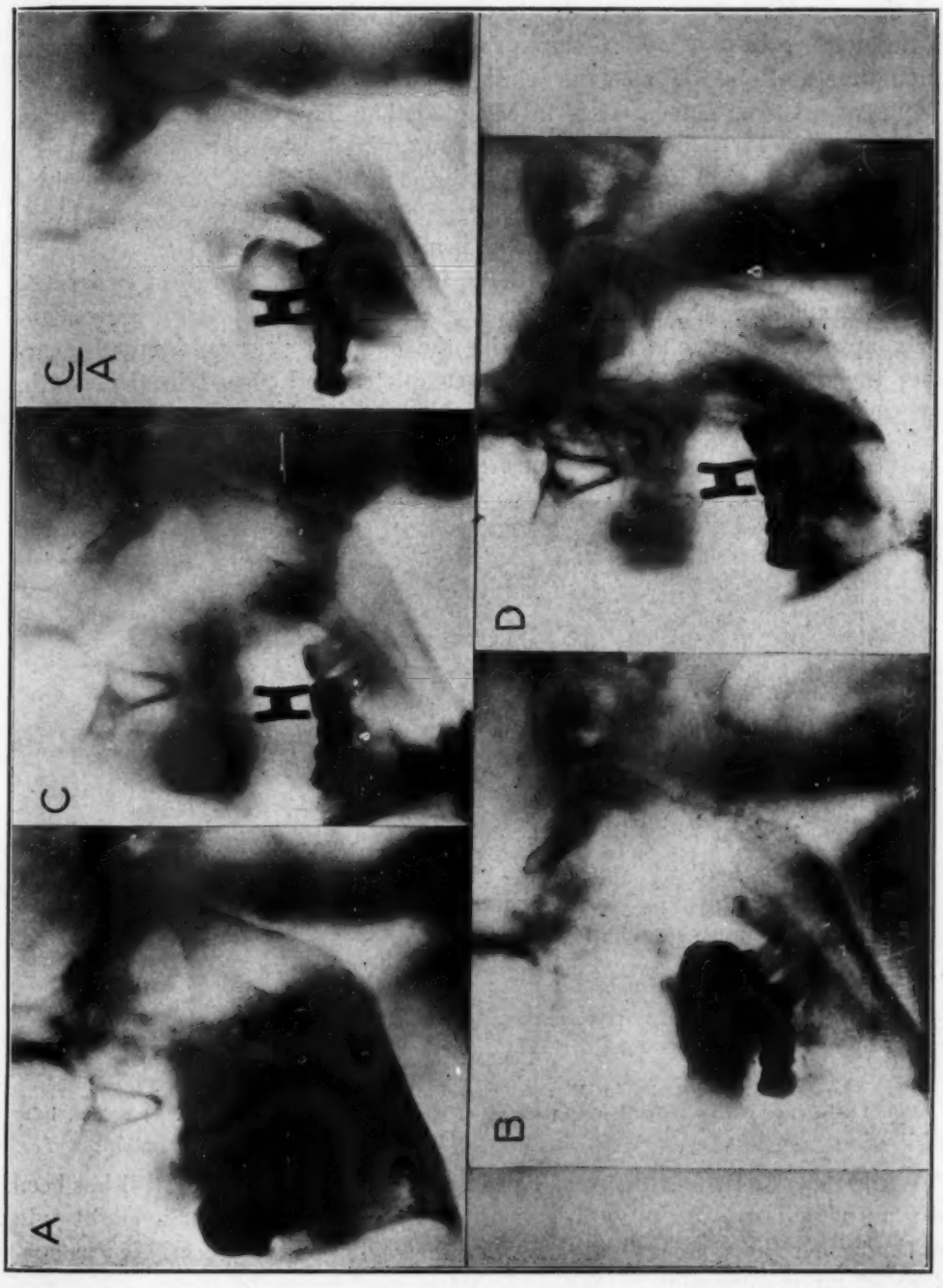


Fig. 9.—Precision laminography of the temporomandibular joint. Condyle changes of less than 1 mm. within the glenoid fossa are registered, using a point on the petrotympanic fissure as an anatomic landmark.

patient was advised to have the maxillary teeth removed. Pre-extraction records were made, and the same maxillary-mandibular relationship was retained. The maxillary process was reduced conservatively. A denture was constructed, balancing the occlusion against his own mandibular teeth. Patient

wore the denture for six months. At the end of this time he complained of tiredness in the jaws. By clinical inspection no overclosure was observed.

Laminagrams were taken (Fig. 9 *A*) at a level of 3.4 cm., with the denture in position and the teeth in occlusion to show the exact position the head of the condyle assumed to the fissure in the glenoid fossa. *B* shows a lateral view of relationship taken at the same depth, when the joint was in a relaxed position with the denture in position. *C* shows the new relationship established between the mandible and maxilla, also the relation of the condyle to the fissure, before the denture was completed. It is to be noted that the position of the condyle head is identical to *B*, in that it is in an unstrained condition, this being the physiologic rest position for this case. *D* is the completed series. Another laminagram was taken as a check to show there was no movement of the patient's head, and also a check upon the accuracy of the procedure followed.

It is to be observed that by superimposing laminagraphic record *C* over *A*, so that the anatomic shadows coincide, the condyle head has been moved anteriorly approximately one millimeter by the external pterygoid muscle, correcting the overclosure. A denture was completed and placed in the mouth. There was little discomfort after a few days.

Fig. 10.

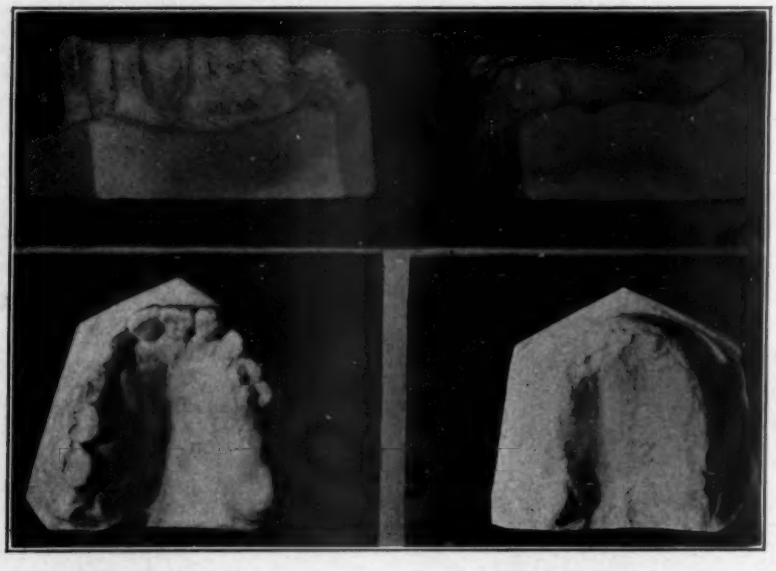


Fig. 11.

Fig. 10.—Case referred to in Fig. 9, right lateral view; before, left; after, right.
Fig. 11.—Conservative reduction of process of clinical case shown in Fig. 9; before, left; after, right.

While there was no evidence of pathologic condition in this joint, it has been shown an overclosure was present, which may have accounted for the slight pain radiating above the zygomatic arch toward the region of the eye. This was suggestive of an involvement of the auriculotemporal nerve by virtue of its distribution to that area. Inasmuch as the condyle showed no direct contact with the petrotympanic fissure, the point at which the chorda tympani nerve leaves the moving condyle (there was no ptyalism), would tend to eliminate the chorda tympani as a causative factor.

While it is not my purpose to present a method of treatment, it is within the scope of this paper to point out the necessity of accuracy in the procedure followed. In the treatment of this case, a point on the petrotympanic fissure taken at a depth of 3.4 cm. was used. Since the laminagraph rays are at right angles to the film and will continue to maintain the depth of 3.4 cm., any changes will be recorded not only in one plane but two planes, an anteroposterior and a mesiolateral.

It may be concluded from the case just presented that (1) pyorrhea may produce an overclosure; (2) referred pains may be caused by a small change in the rest position of the condyle to the glenoid fossa, and (3) condyle changes of less than 1 mm. within the glenoid fossa are demonstrated, standardizing the laminagraphic technique of the temporomandibular joint.

In the cases presented, we have seen the effect malocclusion, when not treated, has had upon the temporomandibular joint in later life, frequently causing pains and much discomfort. It must be remembered, however, we were dealing with the developed bone musculature, and other component parts making up the joint.

In children, the joint as well as the bones of the face are in the process of development and are susceptible to stimulation of changes.

There is a definite relation between the occlusion of the teeth and the position of the head of the condyle within the glenoid fossa when the teeth, jaws, and related parts are completely developed for each individual.

Since there is evidence the glenoid fossa is completely developed between the ages of 13 and 15 years, it would seem any changes made in the position of the condyle prior to this time would be more permanent. However, changes of lesser degree are noted because of the continued development of the bones of the face in a vertical and a horizontal direction.

A roentgen ray study of fetal life was made of the temporomandibular joint and the bones of the cranium, beginning at the eighth week and observing changes, by weeks and months, to and including the first year of life.

It is to be observed that the glenoid fossa and the mandible are present at the sixth month.

This is presented to show definite changes are rapidly taking place which, later in life, will continuously develop into a temporomandibular joint.

Subluxation may be defined as an incomplete dislocation in which the normal relationship of the articular surfaces is disturbed. Extreme subluxation may result in sprains caused by more or less damage to the ligaments, tendons, and muscles comprising the temporomandibular joint.

Dislocations of the condyles of the mandible in children are infrequent even though the concavity of the glenoid fossa is not elliptical, and the articular ementia is not prominent. It is observed in children that in opening the jaw the condyles move forward and slightly downward and rest upon the undeveloped summit of the articular ementia, without displacement, seemingly beyond the point of an adult.

Apparently dislocation is prevented by the position of the coronoid process and the action of the temporal muscle.

Changes of occlusion of the teeth affect the position of the condyle; even though those changes are small, they are present and may be recorded with some difficulty.

It would seem a gradual change of moving the condyles anteriorly would be in conformity with the gradual change of the teeth in the line of occlusion, after the development of the glenoid fossa.

Possibilities of dislocation of the condyles would not likely occur unless the applied force would overreach the physiologic limits of the motion of the joint, or some pathologic changes were present.

CONCLUSIONS

1. A definite relationship exists between the head of the condyle and the glenoid fossa when the teeth are in normal occlusion.

2. Overclosure may be a contributing factor in ear disturbances and referred pains.

3. Symptoms of pain disappear as the function of the joint is restored to normal.

4. To demonstrate definite changes of the condyle within the glenoid fossa, a standardized roentgen ray technique should be used.

5. Precision laminography of the temporomandibular joint has been demonstrated clinically.

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5458 Lillian Avenue

A CASE OF DEEP OVERBITE SHOWING THE PRINCIPLE AND APPLICATION OF THE REMOVABLE BITE PLANE IN TREATMENT

Case Report

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THE value of a case report lies in the opportunity it presents to study the application of certain ideas of concept, principles of treatment, and methods of procedure to some specific condition. Orthodontic treatment is concerned in every instance with the specific problems of some specific individual at some specific time during his growth, development, and adaptation to function.

The human organism is the most complex combination of component factors in the universe and is invariably unique within a very large range of natural variability. This is the starting point from which all reasoning concerning the subject should proceed.

All set rules regarding dimensions and orientation are purely relative and may be very misleading if they divert the mind from the basic nature of the problem.

The fundamental, underlying, and most important single fact to be considered in all orthodontic thought is that it is a biologic process which proceeds to its objectives in a series of coordinated steps, each related to the others but none of them quite predictable.

One of David Starr Jordan's favorite remarks was, "Wisdom consists not so much in knowing what to do in the long run, but what to do next."

An orthodontist is, figuratively speaking, in the position of a field marshal who is constantly facing new situations which require every bit of knowledge, ingenuity, and skill which he possesses, and in which he should use, at the correct time and place, every mechanical means at his command best suited to the immediate problems at hand. The military genius, Napoleon, was well aware of the basic nature of a battle as a biologic problem of mass activity by his well-known saying, "Always expect the unexpected." In biologic processes the unexpected very often becomes the inevitable.

Man has a natural aversion to using his initiative or doing much personal thinking and is always looking for some system to follow in order to avoid this. The profession has had many of them. Some remarks of Abbe Dimnet's regarding the racial philosophy of the originator of one of them might be apt at this point. "The German mentality," he said, speaking of Hitler's hold on the Fatherland, "loves an intellectual frame, even though it is not clothed in fact, which explains the failure of the German plan for the drive on Paris. The Germans

Read before the Thirty-Seventh Annual Meeting of the American Association of Orthodontists, Kansas City, Mo., April, 1939.

Fig. 1.

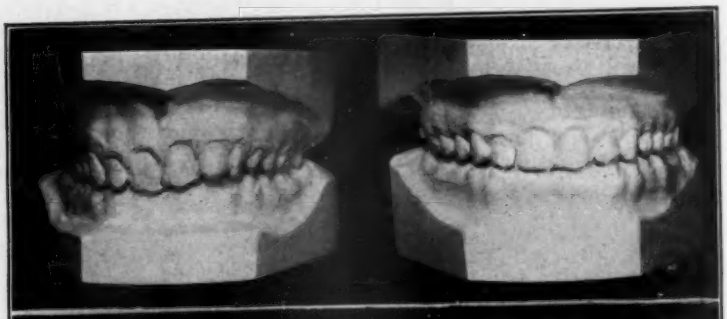


Fig. 2.

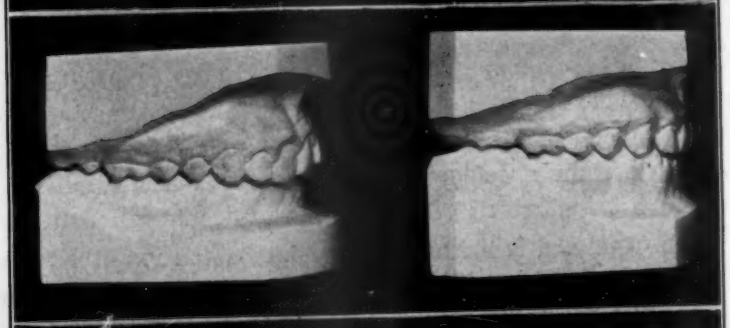
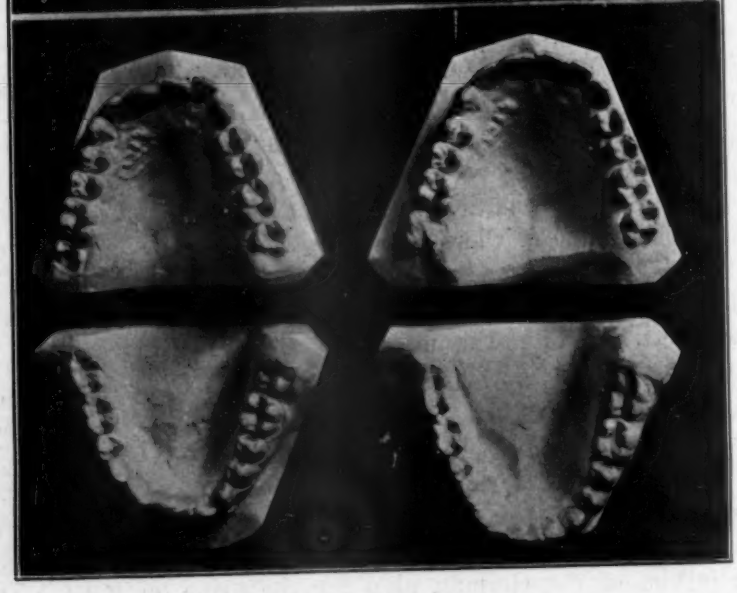


Fig. 3.

Fig. 4.



couldn't accommodate the plan to the facts, to changing conditions. The plan must be right. They left their right flank exposed and drove on to failure."

With these opening remarks as a philosophic background, I have selected for presentation a quite common and ordinary case which I am fully aware can be treated successfully by various methods, but in which the ones I have employed, I trust, contain a modicum of logic and common sense and are based on principles applicable in a broad and fundamental sense to all cases.

Growth and function are ever present during treatment and can, and should, be the greatest allies of the orthodontist; therefore, the least interference he can offer them and the more he can enlist their aid in the natural performance of their functions, the more sensible should be the treatment.

Fig. 5.

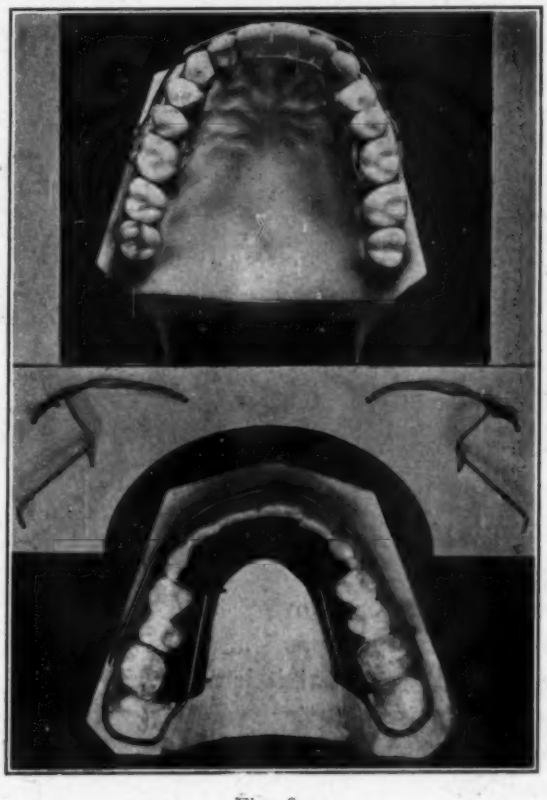


Fig. 6.

Figs. 1 to 4 show a case commonly known as one of unilateral distoclusion with an excessive overbite. Conforming to one line of thought this case might be successfully treated by a method calling for the banding of possibly twenty or even all twenty-four of the erupted teeth and producing the desired changes by manipulations of labial arches exclusively. On the other hand, it might also be successfully treated with only a minimum of appliances combined with properly conducted myofunctional therapy.

Following a line of thought which I have presented in previous papers, that structural balance, functional efficiency, and esthetic harmony are the objectives

of treatment in all cases, these objectives in themselves constitute a natural classification for the major issues and offer a basis for individual analysis.

It is obvious that the first and third of the objectives stated offer no problem in this case, because it is apparent that there is a good proportion of tooth substance to bony development, and that the facial proportions should be quite satisfactory with the full complement of the teeth in their correct individual and cranial relations. The major issue is confined to the second objective.

The simple expedient of directing the patient to occlude the jaws in the correct mesiodistal relation will show in all cases of this kind the exact, specific, and individual causes which interfere with this correct relation. The logical line of reasoning is to proceed to remove the first immediate interference or cause, employing whatever means or appliances are best suited to the particular conditions of the moment, being entirely free to change, if necessary, to some other means once it has accomplished its purpose, or if some untoward reaction is taking place.

Fig. 7. Fig. 8. Fig. 9.

In making the necessary changes in a case of this kind, the mesiodistal relations of the teeth in malocclusion naturally have to be changed, but whether this is actually accomplished by moving teeth bodily through the bone or by changes in the temporomandibular articulation is still a matter of considerable speculation. The probability is that it is a combination of both, but for practical purposes it does not much matter which proportion of each, so long as the final result permits of a correct relation of the teeth and a satisfactory excursion of the mandible.

In treating this particular case it was found that a supraclusion in the incisor region, particularly in regard to certain teeth, was the main interference with the ability to occlude correctly. This brings up the point upon which the selection of appliances has a very important bearing. There are cases in which this supraclusion may be either in the maxillary or mandibular teeth, or in both. It is a sound and proved fact that changes in function will produce changes in form. Appliances which induce these changes are logical in concept and have proved their efficiency by results. Bite planes, guide planes, or whatever other name they may be known by, are among these instruments, but the time, place, and form of their application are again subject to every individual variation of the

case and call for sound judgment in the selection. There are, however, two main forms, each producing a somewhat different result and each applicable specially for a particular condition.

It is quite obvious in this case that the maxillary central incisors and the right lateral incisor are, in varying degrees, the chief objects of interference both in a vertical as well as horizontal relation. A fixed bite plane of the type as illustrated in Fig. 5 will produce the result of guiding the position of the mandible to a new and more satisfactory relation and, at the same time, depress the teeth upon which the prongs rest. This type of bite plane is, therefore, the most desirable one to use in cases of maxillary incisor elongation. It has been my custom for many years to construct these directly in the mouth; this method

Fig. 10. Fig. 11.

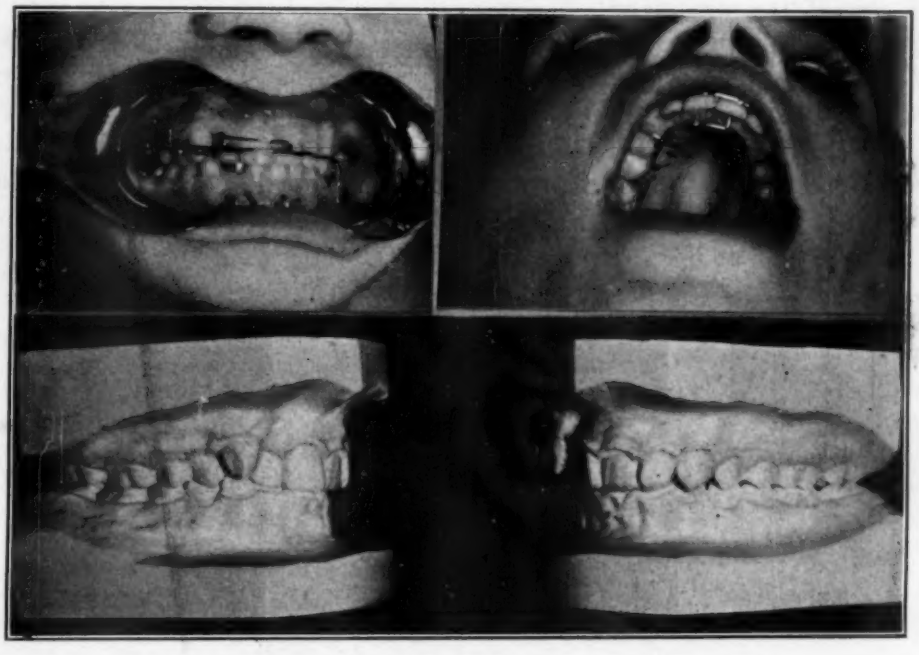


Fig. 12.

permits of great accuracy in locating the prongs at the most advantageous positions and varying their length, depending upon the varying degrees to which it is desirable to depress certain teeth. The use of removable vulcanite bite planes as illustrated in Fig. 6 is indicated, par excellence, in cases in which there is an obvious supraclusion of the mandibular incisors. Both of these types can be used in conjunction with fixed labial appliances when the use of intermaxillary traction is also desired. There is a very subtle but definite distinction in the selection of these two types of bite planes.

The use of the fixed bite plane was employed during most of the active treatment of this case in conjunction with a maxillary labial arch and mandibular lingual arch. The maxillary labial arch was constructed so as to stand away slightly from the incisors to permit of their labial movement, which was produced mainly by the force of occlusion, and to permit of the distal movement of the left molar which carried the anchor band. An examination of the finished

result showed that there was no appreciable tilting distally of the first left maxillary molar which received the intermaxillary elastic force, indicating a strong suspicion that the change which had taken place was mainly a bodily movement of the mandible, with a corresponding change in the temporomandibular articulation. At the present moment the patient is incapable of occluding distally to the position shown in Figs. 7, 8, and 9. The alignment of the mandibular incisors was effected by means of lingual auxiliary springs.

An adjustable bite plane retainer, as shown in Fig. 6, was used to complete the case. It is possible that a shorter overbite might still be desirable, and this can be obtained by building up the plane to permit of a still further improvement in the vertical relation through the natural forces of function.

Figs. 10 and 11 show the application of the same principles and appliances to a case of the same type. The lingual bite-plane arch shows that recurved Crozat springs have been soldered to it to move the premolar distally, after there was an obvious distal movement of the molars as a result of intermaxillary force. The original models of this case are shown in Fig. 12.

It will also be noted that the individual movement of a lateral incisor is being effected by means of a labial auxiliary spring, acting quite independently and without interfering with the molar movement, Fig. 10.

1218 Medical Arts Bldg.

A STUDY OF THE HEREDITY OF THE ANOMALIES OF THE JAWS*

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I. METHODS FOR STUDYING HUMAN HEREDITY

THE following methods are used for studying human heredity:

1. The genealogic-statistical methods;
2. The study of twins;
3. Conclusions from analogy with experimental data;
4. The calculation of correlation.

The last method has not been used before in the study of anomalies of the jaws.

1. *The Genealogic-Statistical Methods.*—The experimental method upon which Mendel founded the science of heredity is not applicable to the human species. Nevertheless, there can be no doubt that all combinations of some practical importance occur as a result of the unions of human beings. Statistics can be compiled concerning the frequency of a given character in groups of kindred individuals and about its mode of manifestation. General conclusions can be reached.

One way of genealogic-statistical investigations is that which is applied to masses of people. It was used recently by Hirosi Iwagaki for the study of what he calls progenia, which includes "mandibular protrusion" and "edge-to-edge bite."

Another kind of genealogic research consists of studying special characters in successive generations of families and in recording the results obtained in pedigrees. A study of heredity according to this method must include at least three generations: the grandparents and their descendants of two generations in the different branches.

The genealogic-statistical methods have played the most important part in establishing what is actually known about human heredity. They are, and will, no doubt, always be, an indispensable foundation of the study of this heredity.

2. *The Study of Twins.*—The genealogic-statistical methods are appropriate for studying the simple modes of heredity, the dominant, recessive, and sex-linked modes, but they are less suitable for the complex modes. In the latter cases, the best method of ascertaining whether a character is hereditary or how far it is a result of environment, is the study of twins. Uniovular twins have the same inheritance. They are identical in all their characters which have their origin in heredity and differ only in those which are determined by environment. Binovular twins differ in the same manner as brothers and sisters who are not twins. They can be concordant in characters depending on heredity, but this concordance is much less frequent here than in uniovular twins. The demonstration of the heredity of a character is given by its decidedly greater frequence in uniovular than in binovular twins.

*Symposium made at the request of the Editor of the American Journal of Orthodontics and Oral Surgery.

†Professor of Orthodontia, University of Ghent.

It is not sufficient to examine a character in a few identical and a few non-identical twins to establish whether it is hereditary or not. The method of studying twins is one of statistics and is based upon the examination of a great number of cases. This eliminates errors which arise from an accidental cumulation of the character or from confusion between uniovular or binovular twins.

Galton's appreciation of the importance of this study of twins for the study of human heredity has, in recent years, gained in popularity. Some authors, by a preference for this method, have been led to underestimate the value of collecting pedigrees.

3. *Conclusions From Analogy With Experimental Data.*—The knowledge of the general laws of human heredity has been founded upon the results of experimental researches on plants and animals; that, for the special characters, almost entirely upon direct observations on men.

Recently, Ritter published the results of experiments on dogs concerning the heredity of morphologic characters of the teeth and jaws. These related to the shape of the denture, the condition of the occlusion, the shape of the teeth, their size, the relation of the size of the teeth to that of the jaw, the existence of supernumerary teeth, and the absence of teeth. This experimental work has in itself a great interest and importance. It brings, moreover, a most valuable control to the conclusions drawn from the usual modes of studying human heredity.

II. THE ELEMENTS FOR THE DIAGNOSIS OF ANOMALIES OF THE JAWS

These anomalies are divided into sagittal, vertical, and transverse, according to their location in a sagittal, vertical, or transverse plane. We carried out particular research in order to determine the part of heredity in the origin of these anomalies in a sagittal direction. According to a nomenclature largely used in France, we call the variations of the jaws in a sagittal plane, prognathism or retrognathism; those in a vertical plane, hypergnathism or hypognathism; those in a transverse plane, endognathism or exognathism. In this paper, also, we use, according to the same nomenclature, the words mesioclusion, distoclusion, anteroclusion.

In the present state of orthodontics, the following methods are used for diagnosis, in addition to the direct examination:

1. Photographs;
2. Plaster models;
3. Measurements;
4. Teleoradiographs.

We did not use the last in our research.

1. *Photographs.*—An extensive use was made of them by Angle. We find reproductions of photographs in most of the modern textbooks of orthodontics, provided with lines, more especially a line indicating the Frankfort horizontal plane, and, in profile, another line marking the fronto-orbital plane of Simon.

There is not, of necessity, an essential difference between photographs and portraits made by artists. This is especially true when the comparison of several portraits of the same person, executed by different artists, allows the control of the accuracy of their work. The portrait of Frederic III (1415-1493), Emperor of Germany (Fig. 1), shows that it is possible to make a diagnosis of the strik-

ing irregularity of this face, just as well as upon the modern photograph of a profile. The presence of a sagittal anomaly is here obvious. By the nomenclature used here, it has to be diagnosed as maxillary retrognathism, with a massive, somewhat prognathous mandible.

It is seldom that so precise a diagnosis can be made by means of portraits executed by artists, for, as a rule, the portraits show three-quarters of the face or are front views. Notwithstanding this fact, sagittal anomalies of the jaws can be noted or be strikingly evident on portraits which are not profiles. There can, for example, be no doubt that something is abnormal in the sagittal relations of the jaws of Maximilian I (1459-1519), son of Frederic III and Emperor of Germany (Fig. 2).

Photographs or portraits made by artists were exclusively used by us for the diagnosis of cases belonging in past generations, which were judged normal or in which the mandible is in forward position in reference to the maxilla. We referred to the last cases in a quite general manner by the name of mandibular prognathism, though some of them may be rather cases of maxillary retrognathism. In the same manner the word mesioclusion is often used as synonymous with Class III, notwithstanding the fact that some of these cases are really distoclusions of the maxilla with normal mandible.

Fig. 1.—Medal of Frederic III (magnified) shows orientation of the portrait according to the Frankfort and orbital planes.

2. *Plaster Models.*—We made, wherever it was at all possible, impressions of the mouths of the persons examined. Plaster models are durable documents concerning the characteristics of the dental arches.

3. *Measurement.*—The best use which can be made of measurement is graphic representation by geometric projection upon an orthogonal system of planes. We only used projections upon a sagittal plane and made the diagnoses in reference to the fronto-orbital plane. The existing anomalies can be determined by comparing the diagrams, obtained in this manner, with types con-

sidered as normal or regular. Many authors have used graphic methods as a help for orthodontic diagnosis.

Fig. 3 is a sagittal diagram of the normal adult around which the head has been reconstructed. Fig. 4 is the same without the reconstruction. The following points are taken for the measurements: the two tragions, the two orbital points, the nasion, the subnasal, the prosthion, the infradental, the gnathion, and the gonion on the left side.

Figs. 18 and 20 illustrate in a striking manner how the diagnosis of sagittal variations of the jaws can be made by the use of our diagrams.

One might object that we have taken as a basis for diagnosis only one diagram of the normal and used it for adults and young persons alike, whereas the normal profile varies and is not the same at all ages.

Fig. 2.—Bernhard Strigel portrait of Maximilian I (extract).

Diagrams published by De Coster, giving the principal cephalometric characteristics at different ages, interesting from the orthodontist's point of view, supply the answer to this objection. This author has shown diagrams of the

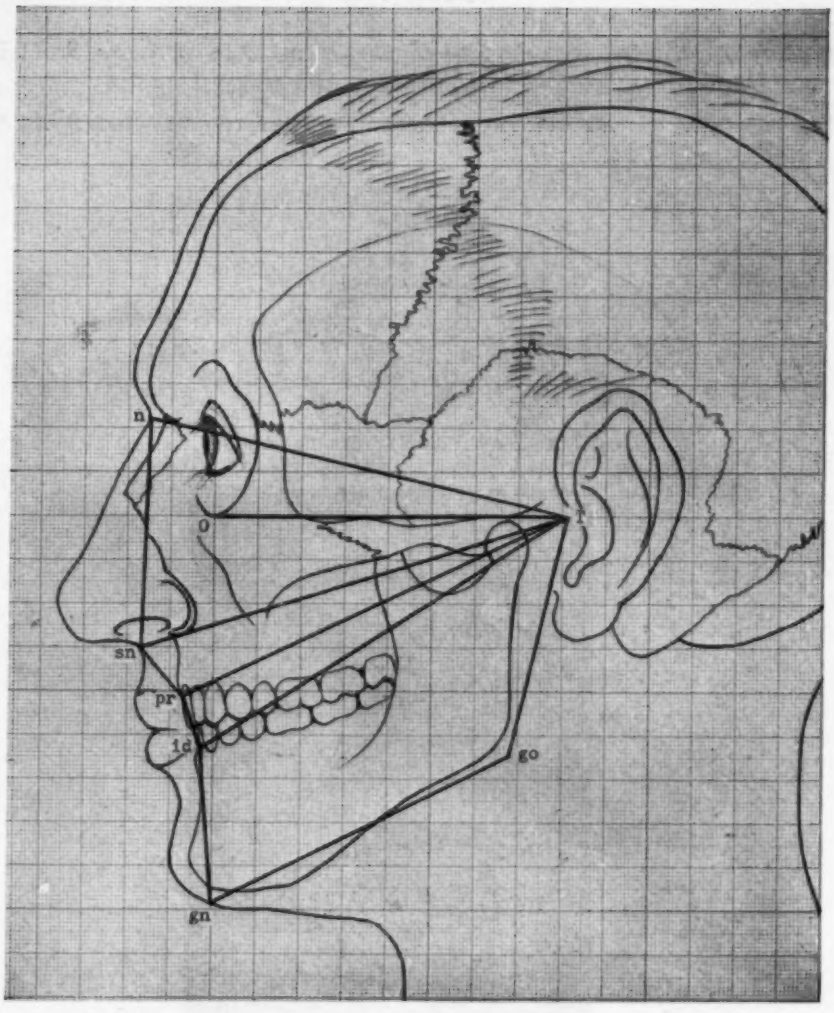

Fig. 3.—Sagittal diagram of the normal adult around which the head has been reconstructed.

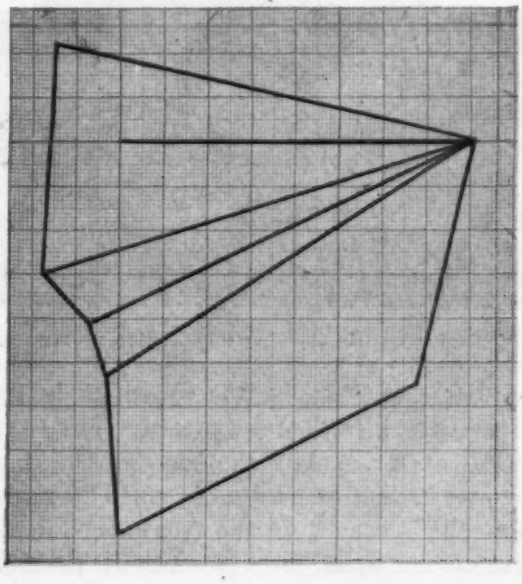

Fig. 4.—Same diagram without the reconstruction.

normal adult, a child of 4, from 6 to 8 years, from 8 to 12 years, from 12 to 15 years, and from 15 to 20 years. Only that of the 4-year-old child differs notably from the others as to cephalometric characters bearing particularly on the subject of this paper.

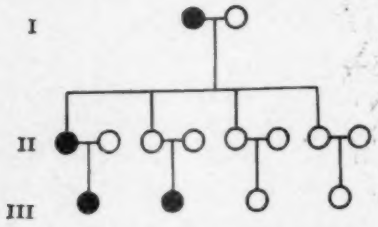

Fig. 5.—Mandibular prognathism.

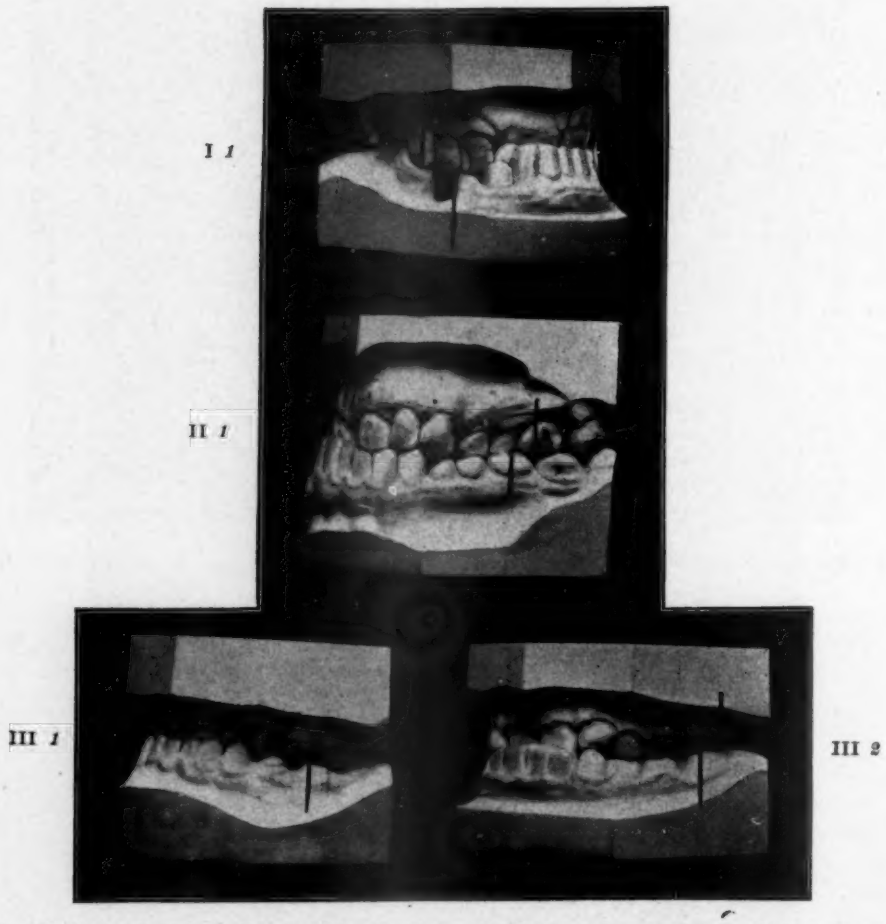

Fig. 6.—Photographs of plaster models showing mandibular prognathism in three generations.

In the present diagram (Fig. 4), the prosthion is 7 mm. anterior to the fronto-orbital plane and the infradental 2 mm. anterior to this plane. The position of these points is approximately the same in the diagram of the normal adult of De Coster, and in that of persons of 15 to 20 years of age. From 6 to 15 years, the infradental is exactly in the fronto-orbital plane. It is necessary to take into account in diagnosis the difference in position of the infradental

noted at this age. From 12 years of age, the position of the prosthion in relation to the fronto-orbital plane in the different diagrams is approximately the same as that of the adult. The variations of the nasion and subnasal points are slight.

III. MANDIBULAR PROGNATHISM AND MAXILLARY RETROGNATHISM

A. PEDIGREES

In all our pedigrees except the fourth, the first number, in each generation, is given to the person who presents the character in the most accentuated manner and the following numbers are given according to the degree of the malocclusion. We do not distinguish between males and females except for pedigree IV.

*Pedigree I** (Fig. 5)

Generation I.—No. *1* is a very accentuated case of Class III, Division 1.

Generation II.—No. *1* belongs to Class III, Division 1. No. *3* has a nearly perfect occlusion.

Generation III.—No. *1*, the teeth are all temporary ones. The mandibular incisors occlude before their antagonists. The mandibular temporary molars present a mesioclusion of nearly 2 mm. *2* is a case of Class III, Division 1.

The four cases with mandibular prognathism present this character at different degrees. It is also worth notice that, in the second line of descendants, the character presented by the grandchild is absent in his parents.

Pedigree II (Fig. 7)

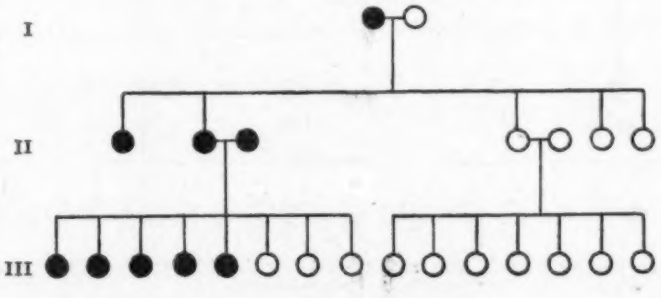

Fig. 7.—Mandibular prognathism.

Generation I.—Our diagnosis for No. *1* is based upon a photograph, which leaves no room for doubt.

Generation II.—We had the opportunity of making a thorough examination of Nos. *1, 2* and *3*, and of taking impressions from Nos. *2* and *3*. These three cases show a very accentuated mandibular prognathism. The teeth still remaining did not permit a more precise diagnosis of the occlusion.

Generation III.—We possess models of the eight first numbers. *1* is a very pronounced case of Class III, Division 1. *2* belongs also to Class III, Division 1. The degree of the anomaly is nearly the same as that of No. *1*. *3* is a case of Class III, Subdivision, almost Division 1. *4*, Class I, the mandibular incisors are in anteroclusion in reference to their antagonists.

The three last numbers of this family have nearly ideal occlusions.

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*We are indebted for this pedigree and the models represented in Fig. 6, to Dr. Lecrenier, Huy (Belgium).

Pedigree III (Fig. 8)

Generation I.—A portrait in oils and a photograph of No. *1* show a very high and prognathous mandible. Our diagnosis for No. *2* is based upon the examination of a good photograph.

Generation II.—We know No. *1* by a photograph, which shows the mandible far exceeding the maxilla. This appreciation is confirmed by the testimony of near relatives. *2.* A photograph shows a heavy and broad mandible and a very developed chin. According to the testimony of near relatives, there was an edge-to-edge bite of the incisors.

Generation III.—No. *1* is an extremely pronounced case of bilateral Class III.

Generation IV.—No. *1* is an extremely pronounced case of bilateral Class III. *2,* bilateral Class III. *3,* Class III, Subdivision. *4.* On each side, the mandibular molars have a mesioclusion of 1 to 2 mm. in reference to the maxillary ones. *5.* On the right side, there is a mesioclusion of 2 mm. of the mandibular molars and premolars with regard to the maxillary ones. *6, 7, 8* and *9* have perfectly regular dentures.

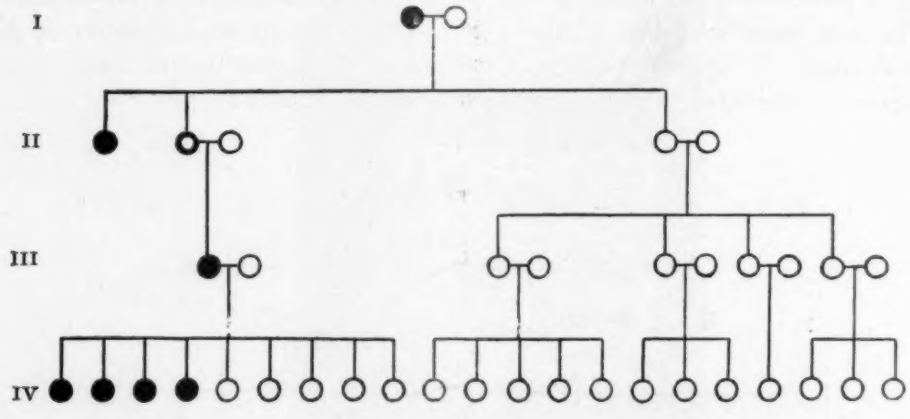


Fig. 8.—Mandibular prognathism. The small circle with thickened outline signifies probable mandibular prognathism.

Pedigree IV

A Pedigree With Nine Generations in the House of Habsburg (Fig. 9)

Mandibular prognathism can be traced in the house of Habsburg, on an interrupted line upon authentic documents, from the end of the fourteenth century until the present time, i.e., during nearly five and a half centuries.

The pedigree here published extends from the end of the fourteenth to the end of the seventeenth centuries. The principal representatives of it are:

Generation I: Ernest the Iron (1377-1424).

Generation II *1:* Frederic III (1415-1493), emperor of Germany.

Generation III *1:* Maximilian I (1459-1519), emperor of Germany. *2:* Mary of Burgundy (1457-1482).

Generation IV *1:* Philip the Fair (1478-1516). *2:* Jane of Castile.

Generation V *3:* Charles V (1500-1558), emperor of Germany. *4:* Isabella of Portugal, his cousin and wife. *7:* Ferdinand I, emperor of Germany.

Generation VI *2:* Philip II, king of Spain (1527-1598). *5:* Anne of Austria, his niece and wife. *6:* Maximilian II, emperor of Germany (1527-1576). *7:* Mary of Austria, his cousin and wife.

Generation VII *4:* Philip III (1578-1621), king of Spain. *5:* Margaret-Mary, his great-niece and wife.

Generation VIII *2:* Philip IV (1605-1665), king of Spain. *3:* Elisabeth of France. *4:* Mary-Anne of Austria, niece of Philip IV and his wife.

Generation IX *4:* Charles II (1661-1700), king of Spain.

Historians and genealogists have been interested for a long time in the special type presented by a very high percentage of members of the House of Habsburg. Brantôme (1535-1614) speaks of the mouth of Charles V in his book: "Les Vies des Dames Galantes."

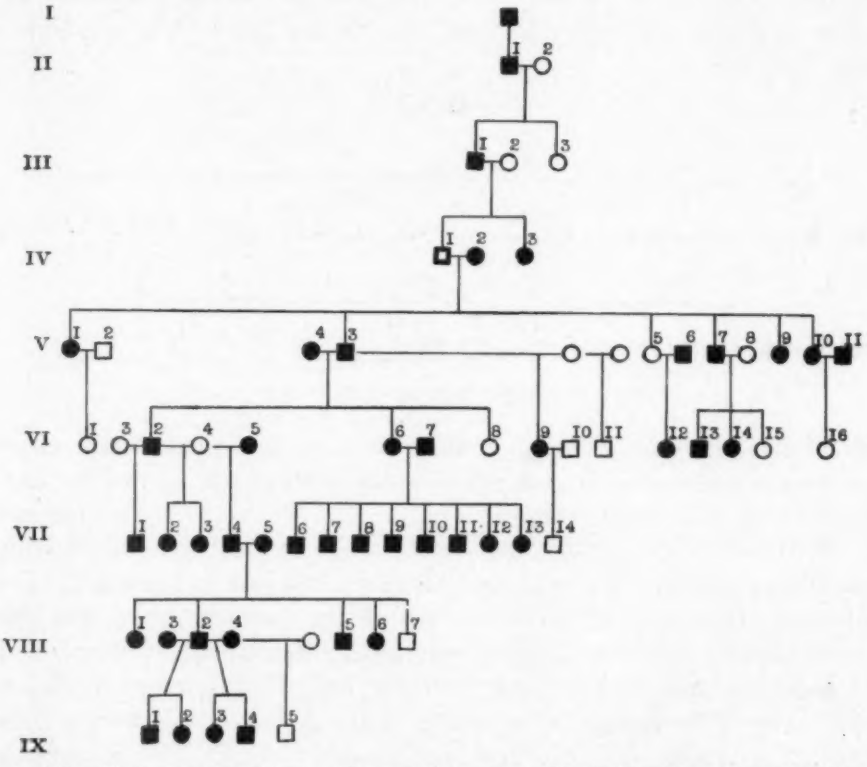


Fig. 9.—Mandibular prognathism. The small square with thickened outline signifies doubtful diagnosis.

A volume was published about the Habsburg family type in 1905 by Galippe, who considered inferior prognathism a sign of degeneracy. In 1910 we published a monograph on the origin of this family type. Other studies on heredity, in reference to this type, were published by Haecker, Strohmayer, Kantorowicz, Mayoral, and Aguilar.

It was not difficult to collect the documents upon which the pedigree of the house of Habsburg, here published, is founded. The three centuries over which these generations are distributed form a period in which art attained one of the greatest efflorescences ever known. Many portraits were painted by official

painters of the sovereigns. Good reproductions of these portraits are to be found in books and articles treating of history, art, or stomatology.

The accuracy of our documentation and conclusions was widely admitted, among others by Fr. Lenz, who reproduced the pedigree, made up by us, in the latest edition of the standard work on human heredity, written by him in collaboration with Baur and Fischer: "Menschliche Erblehre."

Pedigree V (Fig. 10)

The diagnoses of the anomalies in the previous pedigrees are founded upon the sagittal relations of the dental arches, studied in living persons and often also on plaster models or, for past generations, from portraits. In this fifth pedigree and also in the sixth, cephalometric measurements were made of representatives of different generations. In this manner it was possible to make accurate diagnoses of the situation of the jaws in reference to the remainder of the face and to establish whether, and how far, the maxilla or the mandible was abnormal.

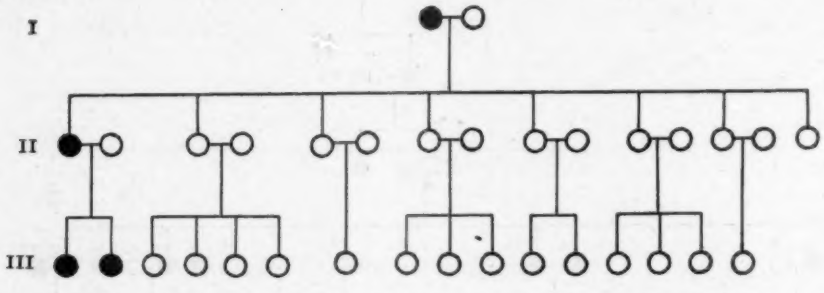


Fig. 10.—Maxillary retrognathism or mandibular prognathism.

Fig. 11 reproduces sagittal diagrams based upon cephalometric measurements on two persons of each generation of the fifth pedigree. To the left are reproduced, from bottom up, diagrams of the son, the mother, and the grandmother. In these three cases, the diagnosis is maxillary retrognathism, with the mandible nearly normal. The son, aged 19 years, is a case of Class III, Division 1. The mother, II *1*, aged 45 years, has lost all her natural teeth. She affirms, in the most explicit manner, that her mandibular natural incisors occluded before the maxillary ones and covered the lower half of their crowns. We made a full set of teeth for her. The relations of the jaws were decidedly those of Class III. The nasion and gnathion are almost normal. The infradental is carried very far back. This position is due to the fact that, in order to place the artificial teeth in correct anatomic occlusion, it was necessary to place the mandibular incisors well back.

The upper and the middle diagrams to the right, of the grandfather and father, respectively, are normal; the lower diagram to the right is that of a girl of 10 years. In consideration of the age, this is a case of mandibular prognathism.

We find in this pedigree superior retrognathism in three successive generations, with a normal mandible. Maxillary retrognathism may consequently exist, while the mandible is normal, and it can be found in successive generations. Moreover, maxillary retrognathism and mandibular prognathism may appear in different members of the same family.

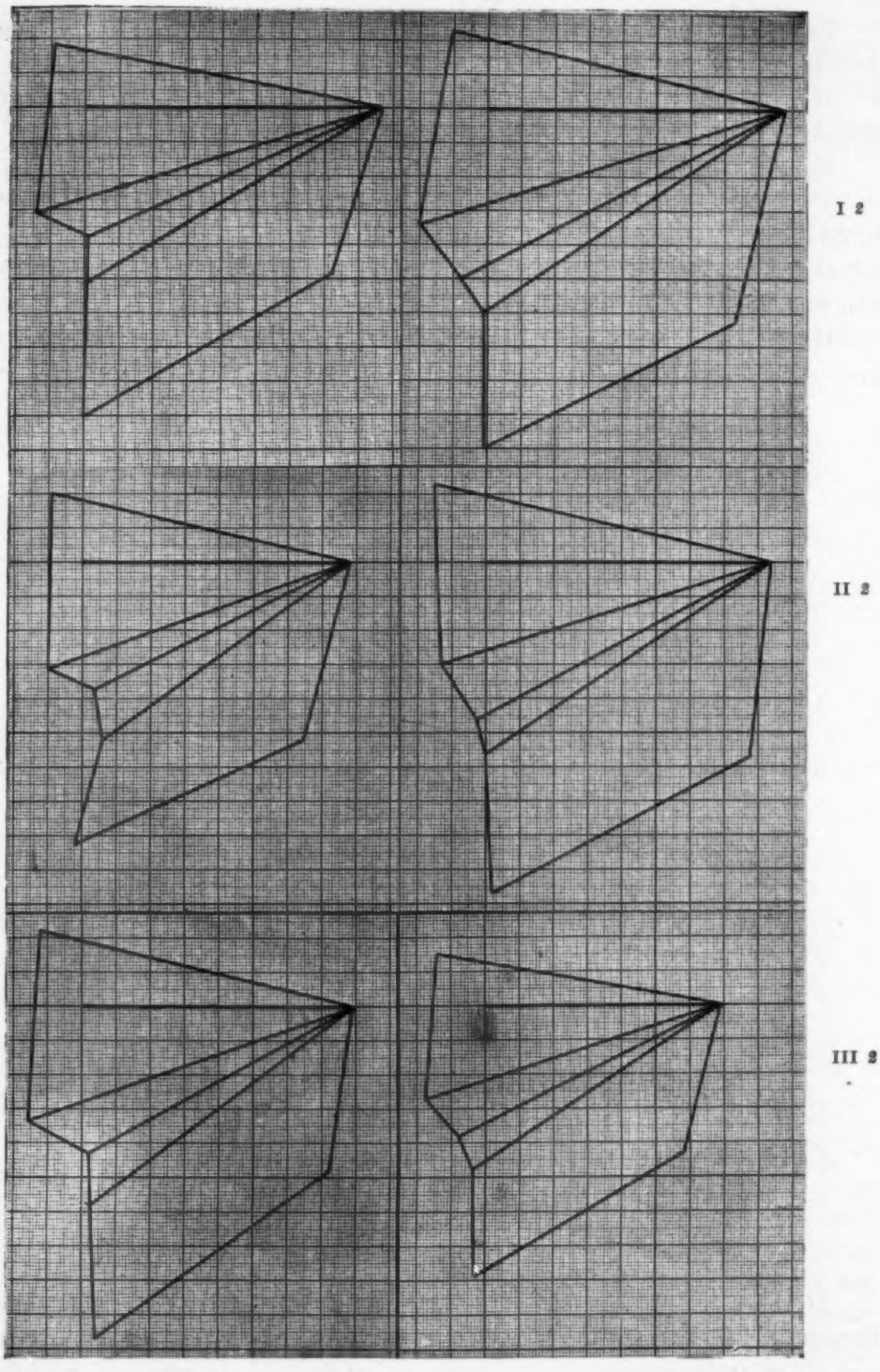


Fig. 11.—Sagittal diagrams to the left show maxillary retrognathism in three generations, III 2, child of 10 years, with mandibular prognathism.

Pedigree VI (Fig. 12)

The diagnosis of *2* of the first generation is made according to a very good photograph (Fig. 13). The diagnoses of *1* and *3* of the second generation and of *3* of the third generation are drawn from diagrams made after cephalometric measurements. Plaster models of these cases were also made.

No. *1* of the second generation (Fig. 14) is a case of Class III, Division 1; $\underline{|6}$ was extracted twenty-five years before, with the consequence that $\underline{|7}$ and $\underline{|8}$ moved forward.

II *3* (Fig. 15) is a case of Class I. This person and the preceding one are sons of a father with mandibular prognathism.

III *3* (Fig. 15), son of II *3*, has Class III, Division 1 malocclusion.

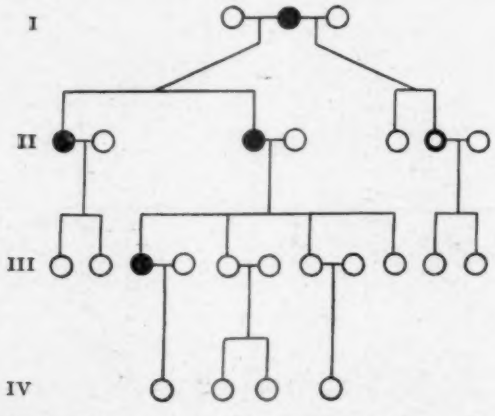


Fig. 12.—Mandibular prognathism or maxillary retrognathism. The small circle with thickened outline indicates probable mandibular prognathism.

Fig. 13.—Photograph of I 2.

If the cases are considered in this manner, the characteristic situation of the mandibular arch, with regard to the maxillary one, has jumped a generation.

If, on the contrary, the diagnoses are established according to the relations of the dental arches in reference to the face, the result is quite different (Fig. 16).

II *1* is a case of light superior retrognathism.

II *3*, the Class I case, presents a very pronounced superior retrognathism. There is also inferior retrognathism.

III *3*, son of II *3*, is a case of light superior retrognathism and is principally characterized by a pronounced inferior prognathism. The discordance between the grandfather, the son II *3*, and the grandson III *3*, has disappeared.

The different aspects of the mandible in II *1*, II *3*, and III *3*, should be explained.

The consideration of a case of inferior prognathism in a child of 3½ years of age throws light upon these differences (Fig. 17). At the moment of closing the mouth, the maxillary and mandibular incisors met edge to edge. By further closure, the whole mandible was swung forward and the mandibular incisors were brought well in front of the maxillary ones. This situation is represented by *A*, Fig. 17.

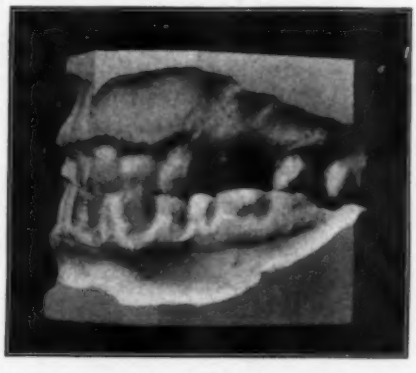
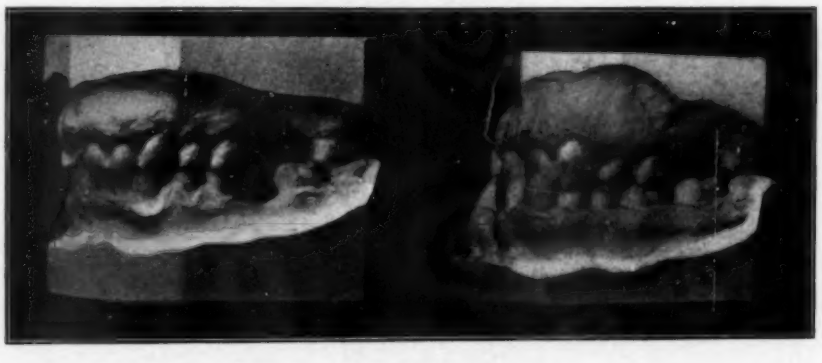


Fig. 14.—Models of II *1*.

II *3*



III

Fig. 15.

Treatment of this case consisted in extensive grinding of the maxillary canines with slight grinding of the mandibular ones and the insertion of an inclined plane on the mandibular incisors and canines. The bite was opened, and the luxation of the mandible, which had in a great measure contributed to establish the situation represented in *A*, did not recur. The result represented Fig. 17, *B*, shows the mandible in a much more posterior position than in *A*. The diagram *A* resembles III *3*, while *B* is near to II *1*.

In the Class I case, II *3*, the bite is low (Fig. 16), and it seems logical to admit that the occlusion of the temporary teeth was the same. The permanent

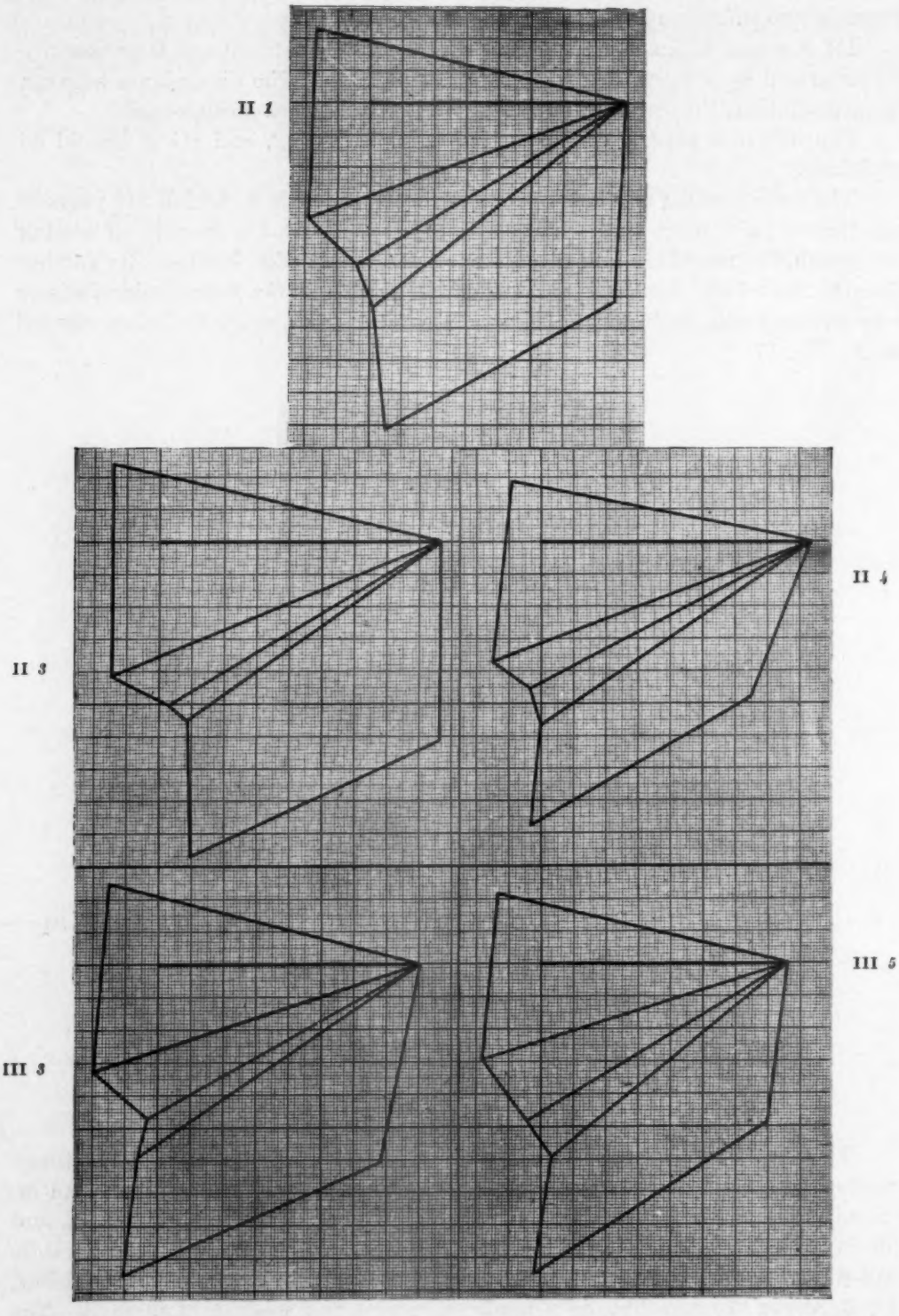


Fig. 16.—II *1*, shows light maxillary retrognathism with nearly normal mandible; II *3*, maxillary and mandibular retrognathism; III *3*, light maxillary retrognathism with mandibular prognathism.

teeth have no doubt erupted in occlusion of Class I and continued to stay in this relation. A low bite of Class I, once established, has a tendency to be maintained. If such an eventuality happens, the whole mandible is obliged to develop behind the position of the maxillary incisors. As the angle of the mandible gets smaller, the direction of the ascending ramus gets vertical or nearly so. II *3* of Fig. 16 shows the realization of such a process of development.

B. INTERPRETATION OF THE PEDIGREES

Are mandibular prognathism and maxillary retrognathism hereditary?

There is no doubt that mandibular prognathism and maxillary retrognathism can develop under the influence of local causes, such as habits, irregularities in the eruption of the permanent incisors, or in the loss of the temporary ones.

The most generally admitted causes are hypertrophied tonsils, adenoids, and rachitis. We investigated whether these diseases may have caused the anomalies reported in the preceding pedigrees and were not able to discover any relation between them and mandibular prognathism or superior retrognathism.

According to researches of Korkhaus, mandibular prognathism affects 5 per thousand of persons who are at least 14 years of age. In the pedigrees I (Fig. 5), II (Fig. 7), III (Fig. 8), VI (Fig. 12) and in two other pedigrees, which we published elsewhere, the proportion in all the affected branches of the pedigrees is about 50 per cent. It is not far from 100 per cent in the pedigree, made up by us, of the House of Habsburg. On the contrary, not one case exists in the nonaffected branches of the families.

It is not possible that the cases were accidentally grouped in this manner. Their frequence and their distribution over a large number of families prove that mandibular prognathism, in its broadest sense, is dependent on heredity.

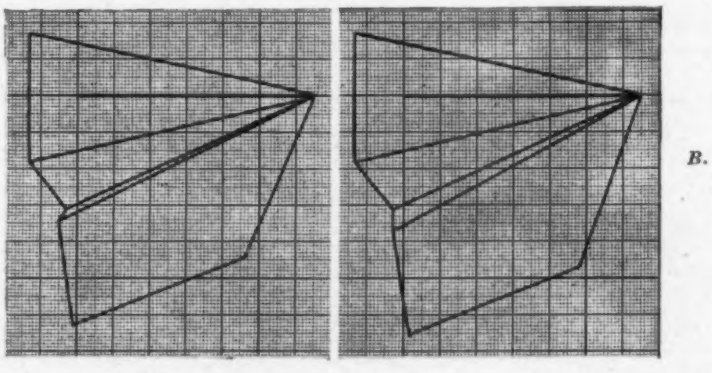


Fig. 17.—Diagram of a child with mandibular prognathism, *A*, on complete closure of the mouth; *B*, after treatment indicated in the text.

A maxilla affected by retrognathism can be traced in successive generations. In other families cases of superior retrognathism and of inferior prognathism, and often intermediary cases between these two types, are found. We did not meet with families presenting only true mandibular prognathism, with normal maxilla, though there is no reason why such families should not exist.

Mandibular prognathism and superior retrognathism are not strictly hereditary, from a purely theoretical point of view, but one or the other appears in a morphologic variation of the jaws which is hereditary.

When development proceeds on what are considered normal lines, the dental arches are placed in normal relations, not only with regard to the rest of the face, but also with each other, in such a manner as to produce what is called normal occlusion.

The hereditary variation on which depend, in a large measure, mandibular prognathism and maxillary retrognathism consists in a retardation of the forward development of the maxilla, in reference to that of the mandible. The mesioclusion of the mandibular dental arch with regard to the maxillary, not hereditary in itself, is in a great measure a consequence of this hereditary change in the relative sagittal position of the jaws.

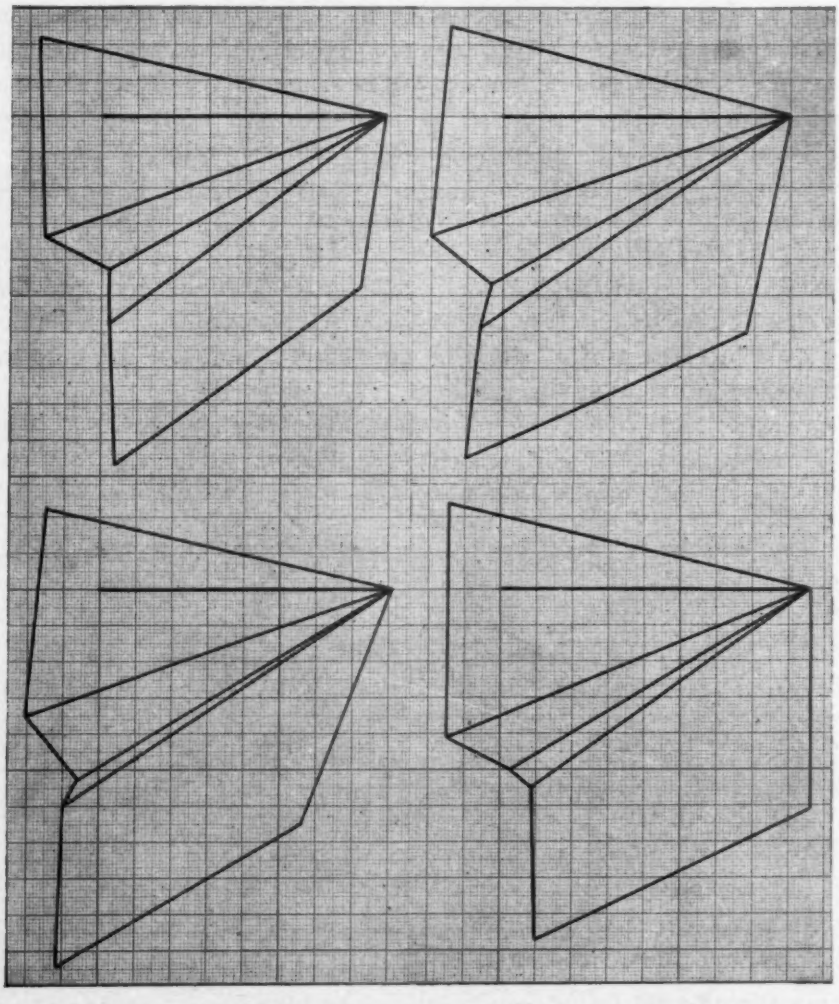


Fig. 18.—*A*, Maxillary retrognathism with nearly normal mandible; *B*, mandibular prognathism with slight maxillary retrognathism; *C*, mandibular prognathism with normal maxilla; *D*, maxillary and mandibular retrognathism.

If the mandible does not exceed anteriorly its normal limit, but the maxilla is in a distal position, there is maxillary retrognathism (Fig. 18, *A*). In other cases, the maxilla may be normal in development and the mandible exceeds its normal limits, that is to say, there is mandibular prognathism without superior retrognathism (Fig. 18, *C*).

In other cases, under the influence of special local conditions, the growth of the mandible anteriorly is impeded, and it may develop backwards abnormally and the clinical aspect of mandibular prognathism does not appear. Heredity seems, in such a case, to jump a generation. It is possible to have an anatomically correct occlusion, with jaws both occupying positions of accentuated retrognathism (Fig. 18, *D*).

Between these extreme cases, there is place for numerous intermediary situations, such as in *B*, where there is true inferior prognathism with slight superior retrognathism.

Exogenous forces may influence these conditions, and in cases of superior retrognathism or inferior prognathism, the mandible is more susceptible to modification than the maxilla. Occlusion plays a considerable part in these changes. At its inception, mandibular prognathism is often due more to a forward swinging of the mandible than to a notable difference between the size and shape of the jaws. The shape of prognathous mandibles depends on the type and the height of the occlusion. General factors may diminish the resistance of the bone and combine with mechanical factors in rendering the angle more obtuse. This explains the multiple aspects presented by prognathous mandibles.

In the two pedigrees, where the diagnosis of the relations of the dental arches to the face was made by means of cephalometric measurements, it was possible to pursue maxillary retrognathism or a combination of maxillary retrognathism and mandibular prognathism in an uninterrupted line. In a case where a link seemed to be missed in the chain of the transmission in each generation, after the diagnosis was made according to the occlusion, it was possible to show that the jumping of a generation was only apparent.

We think that, *in the present state of knowledge, sufficient evidence exists to admit that the factor, on which mandibular prognathism and superior retrognathism depend, is irregularly dominant.*

C. OTHER RESEARCHES ON THE HEREDITY OF MANDIBULAR PROGNATHISM

1. *Pedigrees.*—Downs published (1928) a pedigree including twenty-two persons, in which mandibular prognathism has been observed in three generations. Two extensive, but partially worked out, pedigrees were published by Korkhaus (1931). Keeler published two small pedigrees taken from a study of Hamano (1929).

2. *Mathematical Statistics.*—Iwagaki (1938) collected more than 2,000 family histories concerning mandibular prognathism and examined the hereditary nature of the character from the standpoint of mathematical statistics. The conclusion reached by this study is that mandibular prognathism is hereditary.

3. *Studies on Twins.*—Siemens published a table of cases of mandibular prognathism observed by Hunold, Riepenhausen, Kösters, and himself, and by Praeger. These statistics include four pairs of uniovular twins and six pairs of binovular twins, of whom one partner at least had mesioclusion. Among the four cases of uniovular twins, two were concordant and two were discordant. The six pairs of binovular twins were discordant.

Baker published (1924) a case of two uniovular twins, both affected by mandibular prognathism in nearly identical conditions.

Korkhaus published two cases of uniovular twins, one of which was concordant in reference to Class III, and one discordant, and one case of binovular twins, which was concordant.

Ritter examined 96 pairs of uniovular, 126 pairs of binovular twins and one case of quadruplets. He published models of one uniovular case with concordant Class III occlusion and one uniovular case with concordant anteroclusion of mandibular incisors. Among the binovular twins, there was a pair of girls whose father and grandfather had mandibular prognathism. One of these twins had an extensive mandibular prognathism, the other did not have this anomaly. In another case of discordant binovular twins, one partner presented anteroclusion of the mandibular incisors and in another case, one partner had mandibular prognathism.

From the observations on twins, in reference to the heredity of mandibular prognathism, it is seen that concordance in uniovular twins is much more frequent than in binovular twins.

We must not lose sight of the fact that the above-mentioned studies of twins were made by the simple inspection of the cases, and partly also with the help of plaster models and that the diagnosis was made according to the occlusion of the teeth. We have seen that the variations of the occlusion are not hereditary as such, but depend on a factor, acting upon the size and shape of the jaws. The occlusion gives only an indirect and approximate indication about this more profound action.

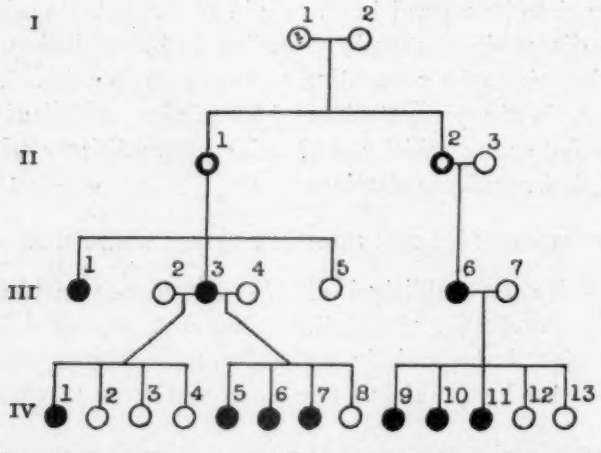


Fig. 19.—Mandibular retrognathism or maxillary prognathism. The small circle with thickened outline indicates probable mandibular retrognathism.

Observations on twins up to the present show decidedly that mandibular prognathism is greatly influenced by heredity.

4. *Experimental Researches on Dogs.*—Ritter crossed a sheep dog of long pedigree with a bulldog bitch of equal pedigree and obtained ten descendants. One of these showed, on the left, anteroclusion of the mandibular incisors; this one was crossed with a bulldog, of long pedigree. Of the nine descendants, one had a denture of a bulldog, one a denture of a sheep dog, two had incisors edge to edge, and five had dentures of a type between the two extremes.

The experimental work of Ritter, the observations on twins and the mathematical genealogic statistics, all kinds of researches pursued by many investi-

gators and upon a most extensive material, confirm, to a degree which can be expected, the conclusions which we have drawn from the study of pedigrees.

IV. INFERIOR RETROGNATHISM AND SUPERIOR PROGNATHISM

A. PEDIGREES

Pedigree VII (Fig. 19)

Generation I.—No. *2* has been judged normal according to a good portrait in oils and the testimony of one of her children.

Generation II.—Nos. *1* and *2*. We have known these persons and examined their mouths and faces. The existing data, though making mandibular retrognathism most probable, were not sufficient to make it absolutely certain.

Generation III.—No. *1* is a case of Class II, Division 1 (Fig. 21).

Fig. 20 shows the diagram and Fig. 21 the models of III *3*. The diagram shows pronounced inferior retrognathism, the infradental being 5 mm. posterior to its normal position. The occlusion is nearly Class II, Division 1. This person married twice. The first wife had a very fine set of teeth, and her father had fine and regular teeth. The second wife has very regular teeth (Fig. 22, III *4*). Four children were born of each of these marriages.

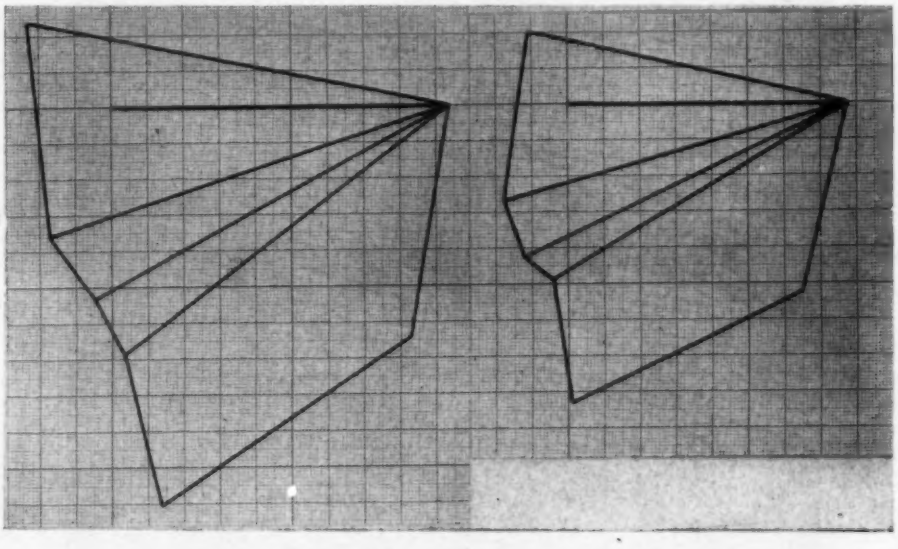


Fig. 20.—III *3*, sagittal diagram of father: mandibular retrognathism. IV 7, sagittal diagram of a son: maxillary prognathism, mandible normal.

Generation IV.—From the children of the first marriage of III *3* (Fig. 21), No. *1* is a case of bilateral Class II, neither Division 1 nor Division 2, the maxillary incisors having a normal inclination. Nos. *2*, *3* and *4* have regular, almost ideal, dentures.

Second marriage of III *3*. Fig. 22 shows, above left, again the models of the father's mouth and, on the right, that of the mother.

IV *5* is a case of Class II, neither Division 1 nor Division 2. It resembles closely IV *1*, child of the first marriage. IV *6* and *7* are cases of Class II, neither Divisions 1 nor 2. IV *8* has an anatomically correct occlusion.

Diagrams of almost all these persons have been made. The cases of Class II of whom diagrams were made are all, except one, cases of inferior retrognathism

with a normal maxilla. There is only one case of maxillary prognathism with a normal mandible (Fig. 20, IV *7*). We have here superior prognathism with normal mandible in the same family as mandibular retrognathism with normal maxilla, just in the same manner as we have seen, in families of the preceding pedigrees, cases of maxillary retrognathism and normal mandible, together with one or more cases of mandibular prognathism and maxilla more or less normal.

Fig. 23 shows the dentures of six persons and Fig. 24 the sagittal diagrams of their jaws. The youngest member of the family is 15 years old.

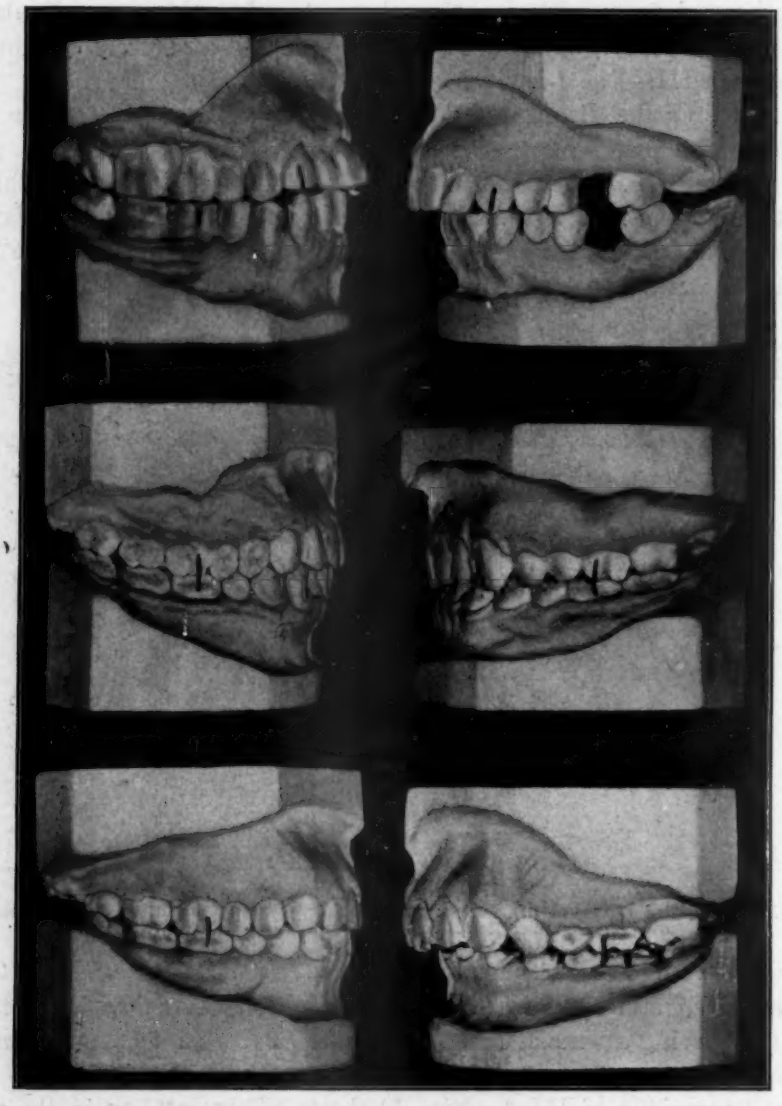


Fig. 21.—Above: models of father and sister of father. In the middle and underneath: models of one child with Class II and of three children with normal dentures (first marriage of III *3*).

III *6* is the first cousin of III *3*. On the diagram, the infradental like that of the cousin (Fig. 20) is 5 mm. posterior to the normal. This is an accentuated case of inferior retrognathism. The father, III *7*, has an anatomically correct denture. One child, IV *13*, has normal occlusion; another child, IV *9*, has Class

II, Division 1 malocclusion; another, IV *10*, has Class II, Division 2; IV *11* is a case of Class II, in which the maxillary arch is approximately normal.

Fig. 19 shows three cases of mandibular retrognathism, on four persons of the third generation: sister, brother, first cousin and, in thirteen persons of the fourth generation, seven cases of Class II: brothers, sisters, or second cousins. In the fourth generation, inferior retrognathism exists in one of the parents of all the members, while the other parent has an almost anatomically correct denture. The proportion of cases of Class II exceeds 50 per cent in the total of all the families of the third and fourth generations.

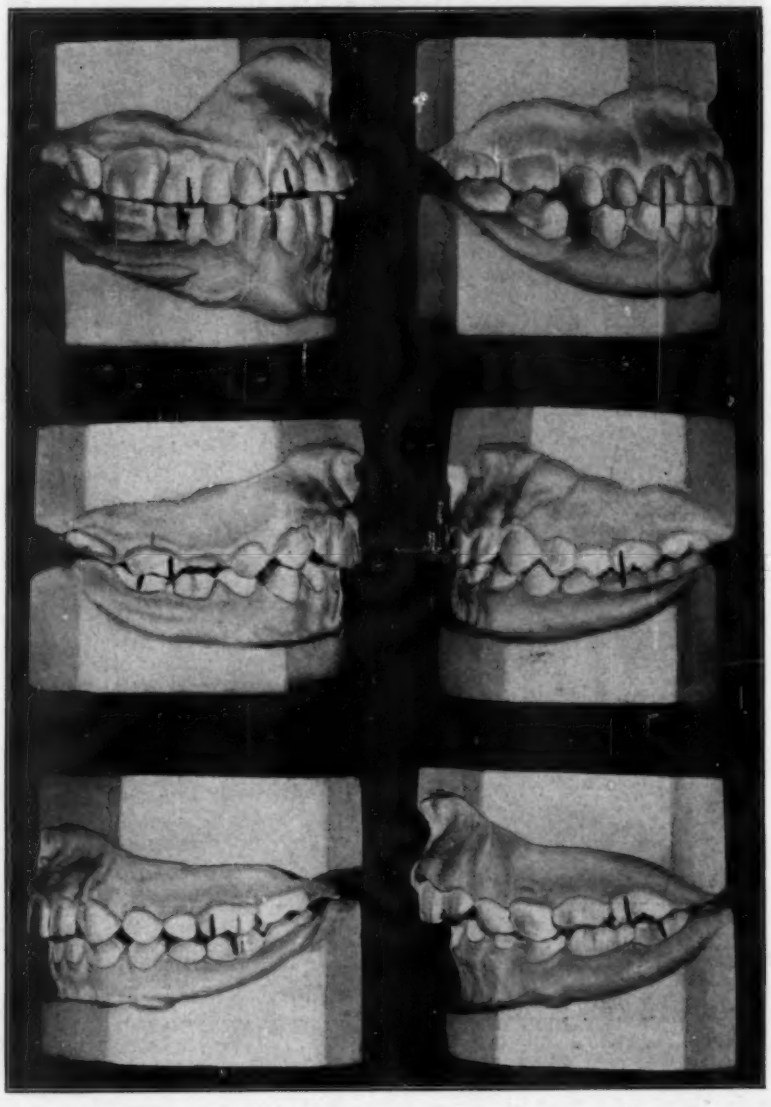


Fig. 22.—Above: models of father, nearly Class II, Division 1. Mother: normal denture. In the middle and underneath: three children with Class II, one with normal denture (second marriage of III *3*).

The chance occurrence of three marriages, in the third generation, of persons with inferior retrognathism to persons with very regular dentures, offers extremely favorable conditions for the evidence of the prospective part of

heredity in the etiology of mandibular retrognathism and maxillary prognathism. If experimentation were possible in this sphere, it would be difficult to find a more favorable concatenation of conditions.

Fig. 23.—Above: models of mother (first cousin of III 3) and father. In the middle and underneath: models of three children with Class II and of one child with normal denture.

B. INTERPRETATION OF PEDIGREES VII AND VIII

Pedigree VIII (Fig. 25)

There is no doubt that inferior retrognathism and maxillary prognathism can have local causes, such as the habit of sucking or biting the thumb, one or more fingers, or the lower lip.

Adenoids are often incriminated. The families of our pedigrees did not show any connection between inferior retrognathism or maxillary prognathism and adenoids. Neither did we find among the cases of our practice any evidence

of such a connection. The Class II, Division 1 cases which we treated and examined in that respect, gave a proportion of 34.6 per cent adenoids. In a detailed cephalometric study of 100 cases of anatomically correct occlusions, Meyer (Basel) ascertained the existence of thirty-three cases of adenoids.

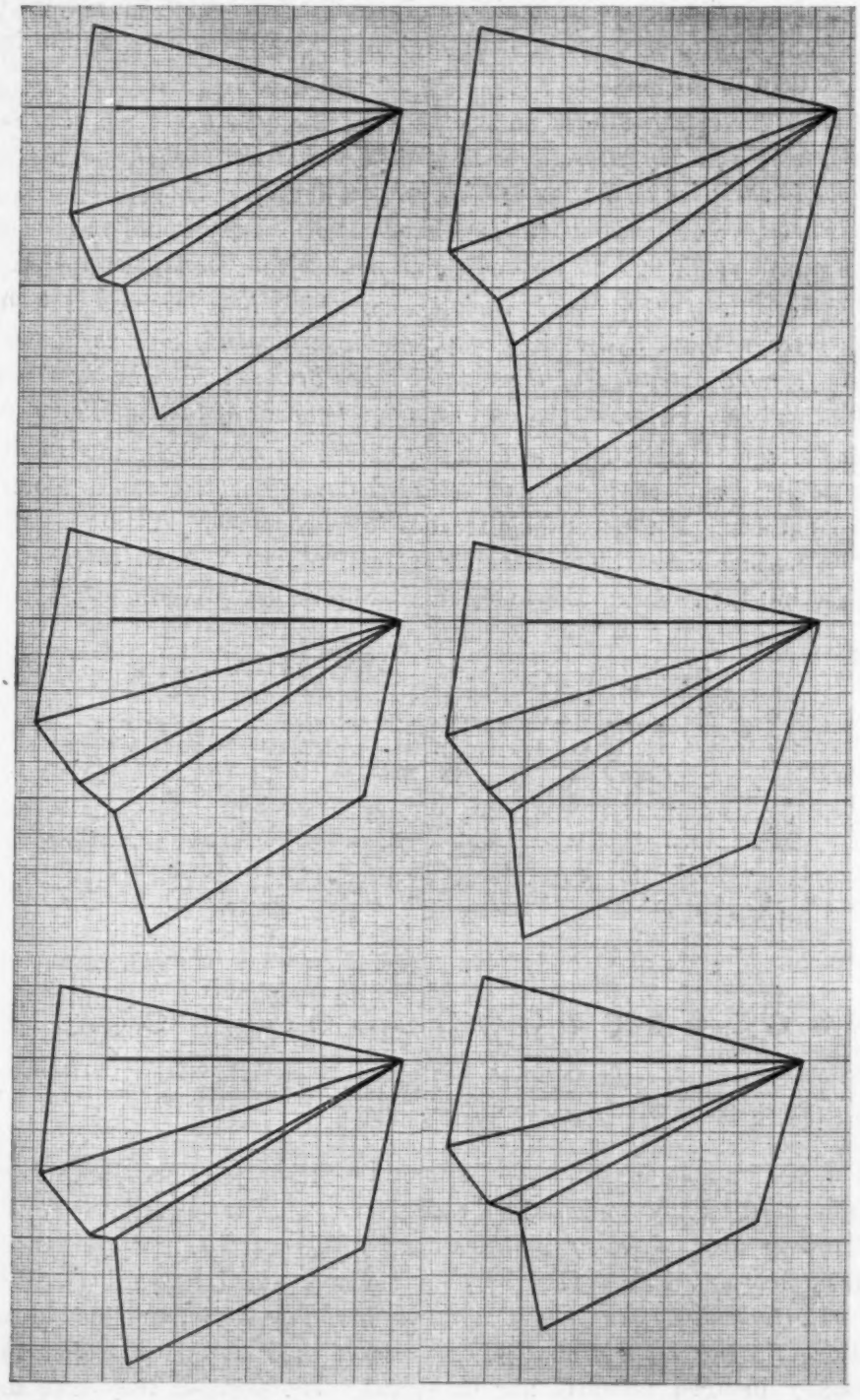


Fig. 24.—Sagittal maxillofacial diagrams of the same persons as in Fig. 23. There are four cases of mandibular retrognathism with maxilla normal and two normal cases.

Our observations concerning inferior retrognathism and superior prognathism agree entirely with those made by us for inferior prognathism and superior retrognathism. It is possible to meet, in successive generations, cases of mandibular retrognathism. This may alternate with maxillary prognathism, or a combination of mandibular retrognathism and maxillary prognathism may be present, or even a retrognathism of both jaws in a more pronounced state in the mandible than in the maxilla.

Mandibular retrognathism and maxillary prognathism are not strictly hereditary, from a purely theoretic point of view, but one or the other appears in a morphologic variation of the jaws which is hereditary.

In the course of development considered as normal, the dental arches occupy relative positions which tend to produce an anatomically correct occlusion.

The hereditary morphologic variation on which, in a large measure, mandibular retrognathism and maxillary prognathism depend, consists in the fact that the forward extension of the mandible has been retarded in the course of evolution and remains so in relation to the development of the maxilla.

Most frequently the maxilla does not exceed its normal forward limit and the mandible, as a consequence of retarded development, occupies a position of retrognathism. If the mandible reaches full development but the maxilla exceeds its normal limit, there is superior prognathism without inferior retrognathism, but the two conditions may be simultaneously present.

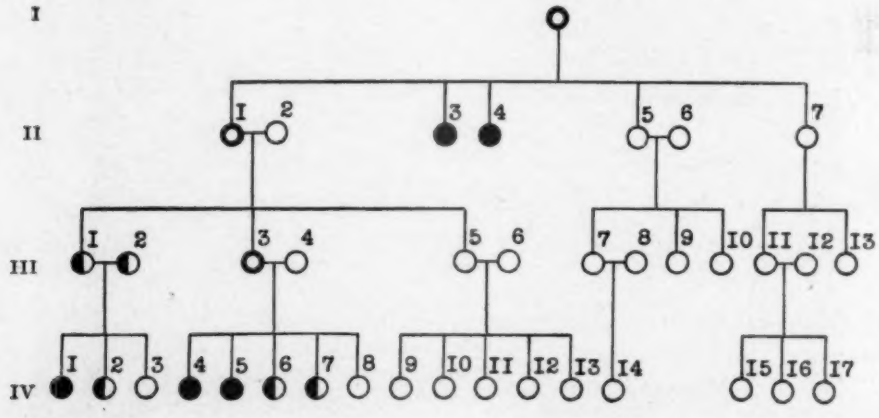


Fig. 25.—The circles with thickened outline indicate probable mandibular retrognathism or maxillary prognathism. The black circles indicate Class II, Division 1 or 2. The circles with left side black signify Class II, Subdivision.

The size and shape of the jaws do not depend only on heredity. They are also influenced by environment. In cases of mandibular retrognathism or maxillary prognathism, three possibilities exist with regard to the position of the maxillary incisors. They may be abnormally inclined forward (position of Class II, Division 1), or take a downward and backward inclined direction (position of Class II, Division 2), or preserve their normal direction, neither abnormally inclined forward, nor backward. The position they take in that respect depends, no doubt, on external causes.

C. STUDY OF TWINS

Siemens published (1928) a table of cases of distal occlusion studied, since 1923, by Hunold, Riepenhausen, Kösters and himself, and by Praeger. The whole of these statistics comprises seventeen pairs of uniovular and sixteen pairs of binovular twins, of whom one at least had a distoclusion. Among the seventeen pairs of uniovular twins, eight were typically concordant, three, atypically concordant, and six, discordant. From the sixteen pairs of binovular twins, one was typically concordant, thirteen, discordant.

A statistical survey published by Korkhaus gives, among twelve pairs of uniovular twins, eight concordances and four discordances and, among eleven pairs of binovular twins, five concordances and six discordances.

The observations of Ritter on 96 pairs of uniovular twins and 126 pairs of binovular twins gave the following results: six pairs of uniovular twins had a distoclusion bilaterally; one pair had a distoclusion on the right and a cusp-to-cusp occlusion on the left. In another pair there was an edge-to-edge occlusion on the left, while in another pair one twin had a bilateral distoclusion and the other, on one side a distoclusion and on the other side a cusp-to-cusp occlusion. Two pairs only of uniovular twins showed a dissimilarity from the point of view of distoclusion.

Among the binovular twins, one pair only were alike; twelve pairs were dissimilar.

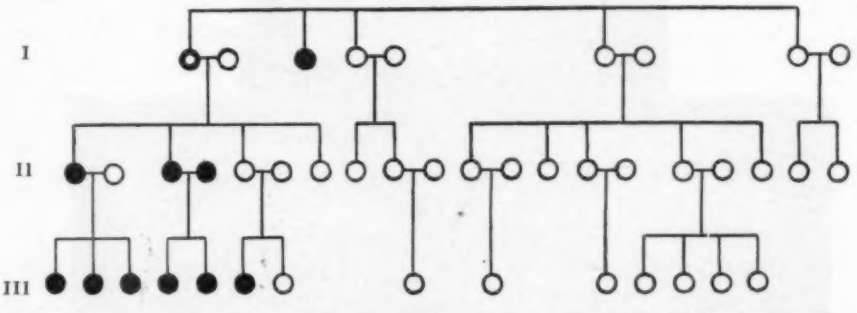


Fig. 26.—Anterosuperior hypergnathism. The small circle with thickened outline signifies probable anterosuperior hypergnathism.

Quadruplets, observed also by Ritter, formed two uniovular pairs. One pair showed concordant distoclusion and the other pair normal occlusion.

Ritter writes that it is not possible to admit that bottle feeding, finger-sucking, or use of the comforter have produced a completely concordant compression of the maxilla in the strongest pair of twins and had no influence on the most feeble pair, the partners of which have broad, perfectly regular dentures. The anomaly must be genotypical.

Ritter agrees with all the conclusions which we have drawn from the study of pedigrees, with regard to the influence of heredity on mandibular retrognathism and maxillary prognathism.

V. THE VERTICAL AND THE TRANSVERSE ANOMALIES OF THE JAWS

1. *Anterosuperior Hypergnathism.*—This morphologic variation is characterized by a very pronounced development of the region corresponding to the maxillary incisors and canines, a somewhat backward inclination of the in-

cisors, which cover completely or nearly their mandibular antagonists, and by a diminution of the height of the occlusion of the lateral parts of the jaws.

The relations of the jaws in sagittal direction are most frequently, in anterosuperior hypergnathism, those of Class I. They may belong also to Class II, Division 2.

Heredity of the anterosuperior hypergnathism has been demonstrated by observations on twins and by pedigrees. Observations on twins, with regard to heredity of anterosuperior hypergnathism, were published by Korkhaus (1928), Siemens (1928), Kösters (1929), Zeiger (1929). Fig. 26 shows a pedigree which we collected. Meier published (1930) several analogous pedigrees. *The pedigrees published up to the present allow the conclusion that very probably the heredity of anterosuperior hypergnathism depends on a dominant factor.*

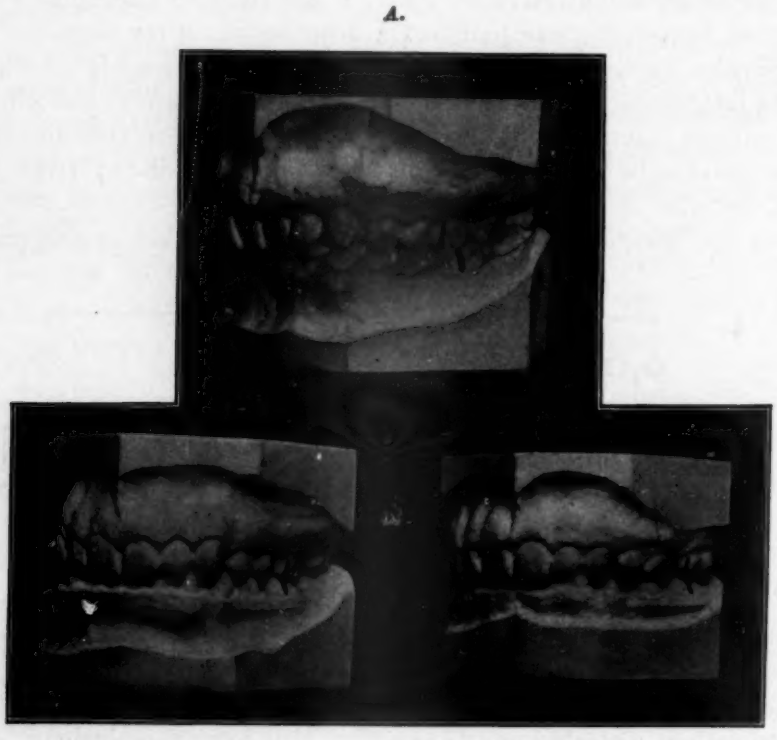


Fig. 27.—Photographs of models with anterosuperior hypergnathism, *A*, father of *B*; *C*, second cousin of *B*.

Fig. 27 shows in *B* and *C* models of the mouths of two second cousins, both with anterosuperior hypergnathism. As the common ancestor from which this type is undoubtedly descended is a great-grandparent, we see here the transmission of a character, with small variations, during a century. Fig. 27, *A* shows the models of the mouth of the father of *B*, but the occlusion is Class II, while that of the son is Class I.

In the anterosuperior hypergnathism, the type of occlusion in sagittal direction is but little influenced by heredity.

2. *Hypognathism (Open-Bite).*—Kösters observed three pairs of uniovular twins, of whom two pairs were completely, and one pair was partially, con-

cordant and one pair of binovular twins which was discordant with regard to open-bite. Ritter found concordance in one pair of uniovular twins, while six pairs of binovular twins were discordant.

3. *Endognathism and Exognathism.*—The diagnosis of these anomalies is founded upon the median plane, determined by the unchangeable posterior part of the raphe. Too great a distance of the lateral teeth from this plane of the raphe is called exognathism, too great an approach signifies endognathism.

Endognathism and the degree of it are often determined by using average statistical values, such as the index of Pont. These methods, most useful in practice, establish a correlation between the width of a group of teeth and that of the jaws, against which serious theoretical objections may be made.

The influence of heredity on the width of the jaws has been studied upon twins.

Zeiger studied the influence of heredity and of environment upon different characters of the maxilla on plaster models of more than eighty pairs of twins. As differences between uniovular twins can only be produced by environment, it is possible to establish, by the study of these, the limits of the variability of some characteristics, under the influence of environment. Zeiger gives the results of his measurements on uniovular twins, in the form of variation curves. The variability of the width of the maxilla in the region of the first molars, in uniovular twins, is given in the following figures:

Difference 0	15 per cent
Difference 0.5	30 per cent
Difference 1.5	36 per cent
Difference 2.5	14 per cent
Difference 3.5	3 per cent
Difference 4.5	2 per cent
	100

These measurements show that, in 81 per cent of all cases, the greatest difference between the width of the maxillae, in the region of the first molars of the observed twins, was 1.5 mm. This width is consequently determined, in a most preponderant manner, by heredity.

In the region of the premolars, the influence of heredity is somewhat smaller, but still very important.

Ritter infers from his extensive researches on twins that, with regard to shape and size, the uniovular twins are most often concordant and binovular twins are very often discordant. *Shape and size of the jaws are rigorously determined by heredity, and not only the normal but also often the abnormal form and size.*

SUMMARY

1. Mandibular prognathism and maxillary retrognathism are dependent on heredity, as a rule; local causes are exceptional.

2. Mandibular prognathism and maxillary retrognathism depend, as a rule, upon a hereditary variation consisting in the fact that the forward development of the maxilla is retarded and remains behind in relation to that of the mandible.

3. The mode of heredity of this variation is irregularly dominant.

4. Mandibular retrognathism and maxillary prognathism depend, as a rule, upon a hereditary variation consisting in the fact that the forward extension of the mandible has been retarded in the course of evolution and remains so in relation to the development anteriorly of the maxilla.

5. The type of the occlusion is, in hereditary sagittal variations, influenced to a great extent by heredity.

6. The characteristics of the divisions of Class II are paravariations, in the mandibular retrognathism and the maxillary prognathism.

7. Anterosuperior hypergnathism is hereditary and is very probably determined by a dominant factor. The cases of Class II occurring in this variation are little influenced by heredity.

8. The shape and the size of the jaws are in great measure determined by heredity. Consequently endognathism and exognathism are also, to a great extent, dependent on heredity.

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THE MANAGEMENT OF ACUTE INFECTIONS OF THE FACE AND JAWS

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AS MANY of the acute infections involving the face and jaws are incidental to dental disease, the oral surgeon is the first to be consulted when treatment is indicated. Because of the extreme potential danger attending even the most trivial infection in this region, the problem of management is very important and often taxes the skill and ingenuity of even the most experienced surgeon. Although the general principles of surgery are applicable in the treatment of all types of infection about the face and jaws, there are certain anatomic features to be considered which alter the details of the treatment to a certain degree.

Coller and Yglesias[6] have done the most outstanding work on infections of the face and neck published in recent years. In the face they have described three potential muscular fascial spaces and one potential visceral vascular space. The muscular fascial spaces they have designated as: the space of the body of the mandible; the masticator space; and the parotid space. The visceral vascular space is known as the lateral pharyngeal space. The muscular fascial spaces are formed by fascia covering muscle and the visceral vascular space by fascia covering viscera. In the face the fascia covering muscles is attached above and below to bone and for this reason infections in anatomic spaces formed by this fascia tend to remain localized to the involved space unless the infection breaks through the confining walls of the space. This is not true of infection in the spaces formed by fascia covering viscera as infection may spread either upward or downward to involve anatomic structures at some distance from the site of the original infection.

Space of Mandible.—The space of the body of the mandible is formed by a continuation of the superficial cervical muscular fascia and the middle cervical muscular fascia along with the free mucoperiosteum overlying the alveolar portion of the mandible. The superficial cervical muscular fascial layer and the middle cervical muscular fascial layer are fused as they pass upward above the level of the hyoid bone but separate at the inferior border of the

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mandible. The outer layer becomes attached to and re-enforces the periosteum overlying the anterior and lateral surfaces of the body of the mandible, and the inner layer becomes attached to and re-enforces the periosteum overlying the medial aspect of the body of the mandible. The free tightly adherent mucoperiosteum overlying the alveolar portion of the mandible completes the space which extends from the symphysis of the mandible to include the third molar region (Figs. 1 and 7). Infections in the space of the body of the mandible, because of the firmly attached fascia, tend to remain localized to the space and do not spread unless they break through the confining walls of the space. An abscess in this space may break into the mouth cavity through the free mucous membrane overlying the alveolar portion of the bone on either the buccal or lingual surface; it may break through to the outside of the face by destruction of the periosteum covering the mandible; or extend to the tissues of the floor of the mouth by breaking through below the mucous membrane. If the infection passes posteriorly, the masticator space may be infected.

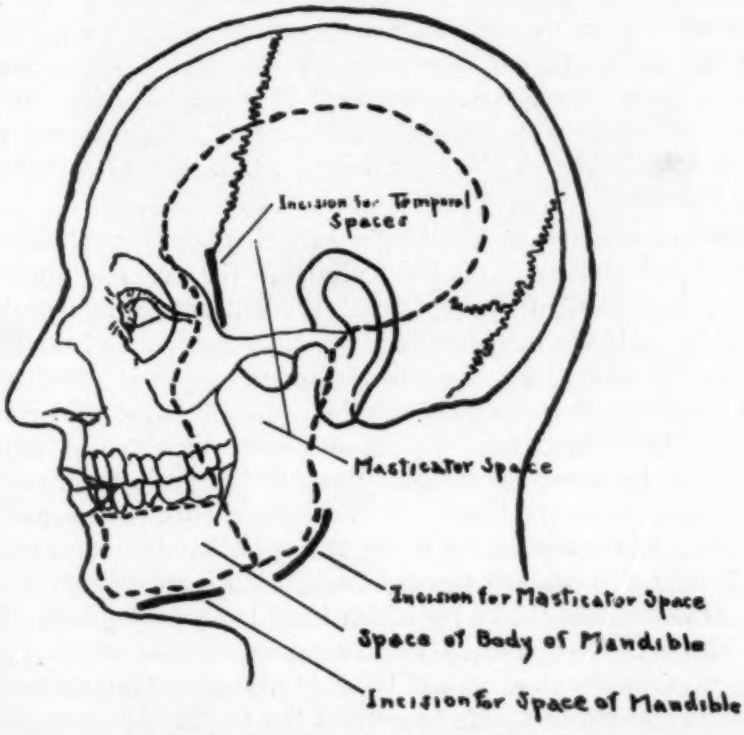

Fig. 1.—Surface projection showing the space of the body of the mandible, the masticator space, and the incisions for drainage of these spaces.

The space of the body of the mandible may be drained by incisions horizontal to the body of the mandible inside the mouth passing through the mucoperiosteum either on the buccal or lingual surface. It may be drained externally by an incision through the skin, subcutaneous fascia and the periosteum of the mandible. The external incision should be below and parallel to the inferior border of the mandible in order to establish dependent drainage as well as to minimize the subsequent scar (Fig. 1).

Dental disease is the most prominent etiological factor in acute infections involving the space of the body of the mandible. The injudicious management of the acutely abscessed tooth, also commonly known as the acute alveolar abscess, acutely infected tooth or "ulcerated tooth" many times results in cervical abscess, osteomyelitis, general sepsis, and not infrequently in death. In any case in which there is pain and swelling about the offending tooth with an elevation of the temperature, pulse, and respirations above normal, it is inadvisable to extract the tooth because of the possibility of spreading the infection with subsequent severe complications. In a series of 234 cases of osteomyelitis of the jaws from various causes Brown and Tung[4] report 105 cases of osteomyelitis of the mandible following the extraction of teeth during the stage of acute infection as against only eighteen when teeth were extracted during the quiescent stage. In this series they also report six deaths following extraction during the acute stage of the infection. In our own clinic we are called upon time after time to reclaim the wreckage resulting from the extraction of teeth during the acute phase of infection. Death in these cases is not an infrequent occurrence. For this reason we urgently recommend more conservative treatment in the management of infections of this type. In our experience we have yet to see a disastrous result following the conservative management of these infections. Instrumentation during the acute stage subjects the tissues to injury over and above that already inflicted by the bacteria with a resultant widespread dissemination of the infection in many cases.

It is our practice first to localize the infection by the application of moist heat intraorally or externally as the findings of the case may dictate. Intraorally heat is best applied by the use of frequent hot saline mouthwashes or throat irrigations, if the infection is in the posterior part of the mouth. Externally massive continuous hot wet dressings are very effective. During the period of localization the pain may be exquisite but can be controlled by the use of opiates if necessary. It has also been suggested by some that the process may be hastened by incising through the periosteum and carefully stripping it away from the bone.[3] This creates a path of lessened resistance and promotes earlier drainage. After the infection has localized, the area should be incised and drained through an incision large enough to insure free drainage. Drainage should be maintained until the acute phase of the infection has subsided. This will be evident by the return of the temperature, pulse, and respirations to a normal level. Only after the acute phase of the infection has subsided is it safe to extract the tooth. After drainage is well established and the general manifestations of infection have subsided, the removal of the tooth may be done at the convenience of the operator.

Masticator Space.—The same layers of fascia that separate at the inferior border of the mandible to form the space of the body of the mandible also form the second potential muscular fascial space known as the masticator space. The superficial layer passes upward externally to the masseter muscle but deep to the parotid gland, Stenson's duct, seventh nerve, and the superficial temporal artery and vein. This layer passes upward over the zygomatic bone, to which it becomes attached, and from there upward over the tem-

poral muscle to become attached to the periosteum of the temporal bone. The middle muscular cervical fascia passes upward medial to the ramus of the mandible to enclose the internal and external pterygoid muscles and becomes attached to the base of the temporal bone. The two layers fuse anteriorly along the anterior border of the masseter and temporal muscles and posteriorly along the posterior border of the ramus and the temporal muscle. The masticator space contains the ramus of the mandible, all of the muscles of mastication, and the fat pad surrounding the attachment of the temporal muscle to the coronoid process (Fig. 1). The upper part of the masticator space is divided into two parts by the temporal muscle. That space between the temporal muscle medially and the superficial temporal fascia laterally is known as the superficial temporal space. The space bounded laterally by the temporal muscle and medially by the periosteum overlying the temporal bone is known as the deep temporal space.

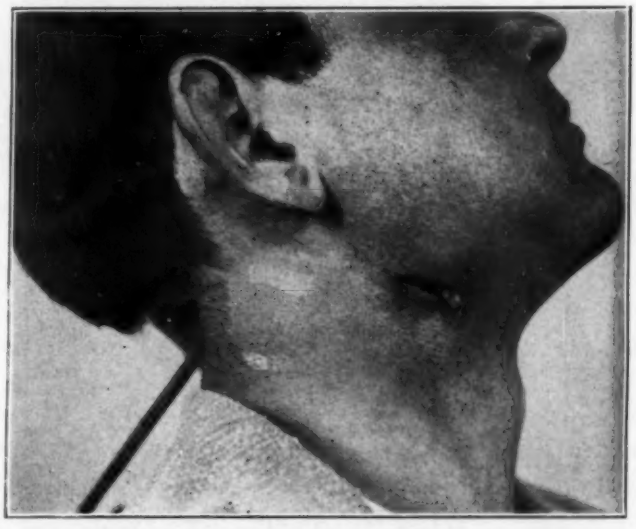

Fig. 2.—Incision for drainage of the lower portion of the masticator space. This infection followed the extraction of an acutely infected mandibular right third molar.

The masticator space may be infected by extension of infection from the space of the body of the mandible, from the parotid space, from the lateral pharyngeal space, or from suppurative middle ear diseases. It is occasionally infected by septic material carried on the point of the needle during the course of injection to anesthetize the inferior alveolar nerve. Infections in the lower portion of the space cannot easily extend downward because of the firm attachment of the periosteum to the bone about the angle of the mandible. The fused layers forming the anterior and posterior boundaries of the space also discourage extension either anteriorly or posteriorly. The infection may spread upward to involve either the superficial or deep temporal spaces. These spaces are also sometimes infected due to osteomyelitis arising in the zygomatic bone or in the temporal bone. Occasionally an abscess in the masticator space may point intraorally, in which event the space may be drained by a vertical incision inside the mouth along the anterior border of the masseter muscle. Through this incision, by blunt dissection, either the portion

of the space lateral to the ramus or that portion of the space medial to the ramus may be explored. The space also may be drained through an incision about 4 cm. long passing through the skin and subcutaneous fascia in a curved fashion below and behind the angle of the mandible (Figs. 1, 2, and 3). Through this incision it is possible by blunt dissection to explore that portion of the masticator space either medial to or lateral to the ramus of the mandible. In cases of extensive infection where either the superficial or deep temporal spaces are also involved, through-and-through drainage should be established between the incision made to drain the lower part of the space and the incision made for drainage of the superficial and deep temporal spaces. The temporal spaces are drained through an incision made vertically through the skin,

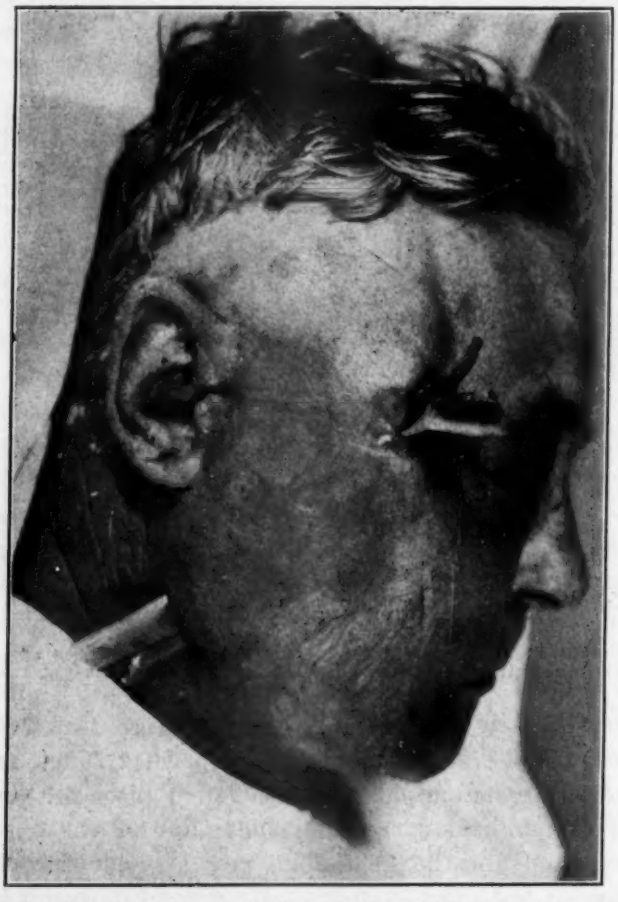

Fig. 3.—Case showing extensive infection of the entire masticator space including the superficial and deep temporal spaces. One of the drains passes medial to the ramus of the mandible, the other lateral to it. The entire ramus of the mandible and the bone of the zygomatic arch were lost due to osteomyelitis. Onset three days following extraction of an acutely infected mandibular right third molar.

superficial fascia and temporal fascia passing upward a distance of 2 to 3 cm. from the angle formed by the junction of the frontal and temporal portions of the zygomatic bone. Through this incision both the superficial and deep temporal spaces may be explored and drained (Figs. 1 and 3).

Severe pain with swelling, difficulty in swallowing, marked trismus and severe constitutional symptoms are early findings in infections involving the

masticator space. Drainage of the masticator space should not be instituted until it is felt that there has been localization of the infection. Usually localization does not take place for five to seven days following the onset of symptoms. Hot wet dressings and general supportive treatment should be instituted to promote localization of the infection.

Parotid Space.—The parotid space is bounded medially and anteriorly by the fascia forming the external boundary of the masticator space and also by the fused superficial and middle cervical muscular fascial layers as they pass backward to enclose the sternocleidomastoid and trapezius muscles. Laterally the parotid space is bounded by the subcutaneous fascia. The parotid space contains the superficial and deep portions of the parotid gland, Stenson's duct, and a portion of the seventh nerve. The parotid space may be involved by extension of an infection through the lateral boundary of the masticator space; by extension from the lateral pharyngeal space; by infections breaking through from the middle ear or mastoid, and by infections carried by the hematogenous and lymphogenous routes. Acute infections in the lateral wall of the pharynx may extend through the fascial layer separating the lateral pharyngeal space from the parotid space and give signs and symptoms of those simulating acute septic parotitis. The parotid space may also be infected directly from an infection of the face extending backward along the course of Stenson's duct. Acute septic parotitis frequently follows extensive operations on the genitourinary tract and the lower bowel in patients whose general resistance is poor. Rankin and Palmer[10] have demonstrated a very definite increase in the incidence of acute septic parotitis coincident with the development of surgical technique permitting more numerous and more extensive operative procedures on the lower portion of the intestinal tract. There are several theories regarding the spontaneous occurrence of acute septic infections of the parotid gland, but the two leading schools of thought are those advancing the theory of infection by the hematogenous route and those advancing the theory of infection by way of direct extension along the mucosa lining the parotid duct. It is felt by some[6] that manipulation over the parotid gland by the anesthetist during the course of a general anesthetic may play an important part in the spread of the infection from the mouth to the gland. It has been noted that as a postoperative complication septic parotitis most always appears in dehydrated malnourished individuals with some degree of sepsis of the oral cavity. In all probability the parotid gland may be infected by either the hematogenous route or by direct extension of the infection from the mouth. As a prophylactic measure we recommend putting the patients in the best possible condition of fluid balance and brushing the teeth every hour with a soft brush and peroxide the day before the operation and again immediately before leaving for the operating room.

A diagnosis of acute septic parotitis is made by finding pain, tenderness, and swelling of the parotid gland along with an increase over normal of the pulse, temperature, and respirations. The infection has an acute onset and runs a rapid course. The constitutional reaction to the infection is quite severe. Treatment should be instituted immediately after the diagnosis is made. Because of the unusually high mortality in this condition watchful

waiting has no place in the management. Treatment with x-ray or radium by means of a radium pack has shown such favorable results in the hands of Rankin and Palmer[10] and DesJardins[7] and in our own clinic that it should be used without fail, immediately after the diagnosis has been made. Adequate fluids are essential and should be given intravenously if there are signs of dehydration. Continuous hot wet dressings applied over the area of the gland are helpful and blood transfusions given to increase the antibody reaction of the blood are of unquestionable value. The normal flow of saliva from the gland should be encouraged by giving the patient lemon juice, lemon candies, or sour foods as a stimulant to secretion. If it is found that the opening to the duct is not patent, an attempt should be made to pass a probe along the course of the duct to be followed daily by dilators. If no improvement in the general condition is noted at the end of forty-eight hours after the onset of the disease and there is no evidence of a return of the temperature, pulse, and respirations to normal, conservative treatment can no

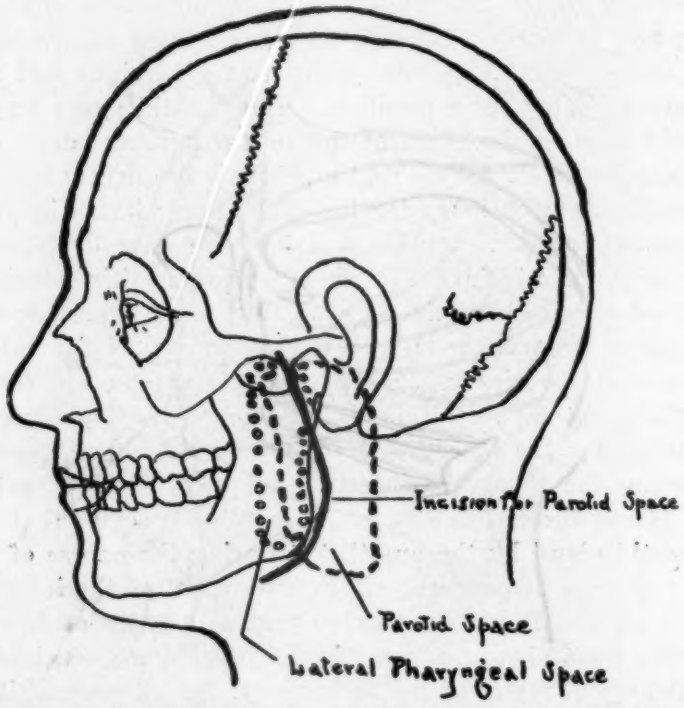

Fig. 4.—Surface projection showing the parotid space and the lateral pharyngeal space. The Blair incision for drainage of the parotid space is outlined. The lateral pharyngeal space may be drained externally through this incision by retracting the parotid gland.

longer be maintained. Incision and drainage should be done without hesitation. The parotid gland is best drained by the incision described by Blair.[2] This incision begins about 1 cm. in front of the tragus of the ear and passes obliquely backward and downward to pass the lobe of the ear, behind the posterior border of the mandible and then around the angle in a curved downward and forward direction for a distance of about 2 cm. below and anterior to the angle of the mandible (Figs. 4 and 5). This incision is made with one sweep of the knife and is carried through the skin, subcutaneous fascia, and the capsule of the parotid gland. As the facial nerve in this area lies between

the superficial and deep portions of the parotid gland, there is little danger of severing the nerve if the incision is carried only through the capsule of the gland. Through this incision both the deep and the superficial portions of the gland may be explored. This is done by blunt dissection. To adequately drain the gland all portions of the capsule must be torn as the individual lobules of the gland are surrounded by dense fibrous connective tissue extending inward from the capsule. The capsule and its septa are so dense that localization of the infection is discouraged, and the patient may die of general sepsis while the surgeon is waiting for evidence of fluctuation in the gland. After incision and drainage the hot wet dressings and general supportive treatment with transfusions given every other day are continued until the infection is under control.

Those cases in which infection is secondary to obstruction of the duct by a stone are of a more chronic nature and usually do not exhibit the symptoms of an acute infection. These cases usually also give a history of painless enlargement over the surface of the gland following meals with disappearance of the swelling a few hours later.

Fig. 5.—The incision for drainage of the parotid space should be made to conform to the posterior border of the ramus of the mandible. This gives the minimal amount of scarring. The figure shows a patient three months following incision for drainage of the parotid space.

Lateral Pharyngeal Space.—The lateral pharyngeal space is bounded anteriorly by the fascia covering the masticator space. The fascia of the parotid gland and the masticator space bound it laterally. Posteriorly it is bounded by the carotid sheath and medially by the fascia of the pharynx (Fig. 4). This space is of only academic interest to the oral surgeon but should be kept in mind because of the possibility of extension of infection from any of the previously mentioned spaces into the lateral pharyngeal space. This space may also be infected from acute infections of the pharynx and from infections involving the floor of the mouth. Infection in this space may readily spread

to the carotid sheath and cause erosion of the carotid artery or septic thrombosis of the internal jugular vein. The infection may also spread from this space to the visceral vascular space of the neck and pass without interruption through the neck into the mediastinum. The lateral pharyngeal space may be drained internally through the lateral pharyngeal wall or externally through the incision described for drainage of the deep portion of the parotid gland.

Sublingual Spaces.—Infections involving the tissues of the floor of the mouth and commonly grouped under the general heading of Ludwig's angina or deep cellulitis of the neck can be approached in as logical a manner as those infections in other parts of the face. The high mortality attending this type of infection has caused it to be one of the most respected and feared of all of the infections of the face. The floor of the mouth is anatomically divided into two large potential fascial spaces each of which is divided into two parts by a median septum composed of dense fascia. The deepest of

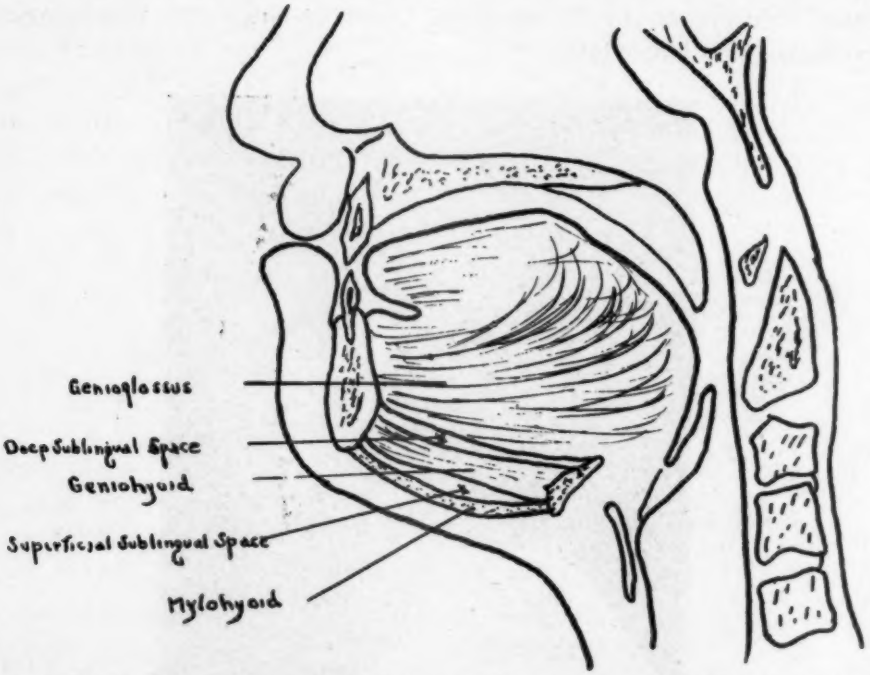

Fig. 6.—Midsagittal section showing the superficial and the deep sublingual spaces.

these sublingual spaces is that space between the genioglossus and the geniohyoid muscles. It is bounded laterally and anteriorly by the lingual surface of the mandible and posteriorly by the hyoid bone. The most superficial of the sublingual spaces is that between the geniohyoid and the mylohyoid muscles and is likewise bounded anteriorly and laterally by the inner surface of the mandible and posteriorly by the hyoid bone (Fig. 6). Each of these spaces as previously described is divided into two parts by the median septum (Fig. 7). Most of the infections in the floor of the mouth involve one or more of these potential spaces. The term deep cellulitis as differentiated from Ludwig's infection no longer has a place in the description of infections in this area. The literature attempting to differentiate deep cellulitis from

Ludwig's angina is voluminous and of no value as these infections are one and the same and the treatment does not vary. The sublingual spaces may become involved by infections extending from the space of the body of the mandible, by infections of the lower lip reaching the spaces by the lymphogenous route, or by infections involving the floor of the mouth and submaxillary gland. The diagnostic features of Ludwig's angina are: pain, tenderness, swelling, and boardlike hardness in the floor of the mouth; acute inflammation of the mucous membranes of the tongue with displacement of the tongue upward and to the side opposite the site of the infection; difficulty in swallowing, chewing, and breathing; excessive salivation with drooling from the mouth; marked elevation of pulse, temperature, and respirations; swelling in the neck underneath the mandible; and a gutteral quality of the voice. General symptoms are severe and discomfort is marked and obvious when the patient is first seen. The localization of the infection usually is difficult to determine. If the most superficial space alone is involved, the major portion of the swelling will be found underneath the mandible, whereas, if the deeper space is involved most of the swelling will be in the floor of the mouth, causing displacement of the tongue (Fig. 8). Early in the course of the disease the infection may be localized to one side, but within a short time it involves both sides of the neck so that the exact localization

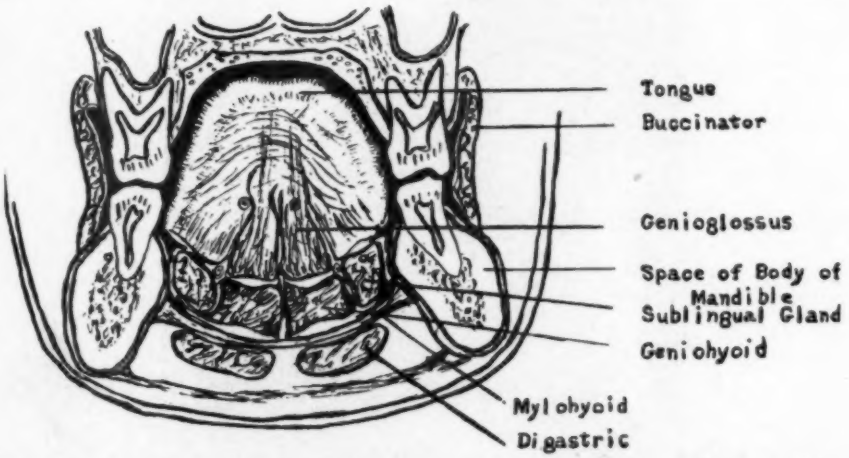

Fig. 7.—Schematic drawing of frontal section of the lower part of the head showing the space of the body of the mandible and the potential fascial spaces in the floor of the mouth. The deep sublingual space is above the geniohyoid; the superficial sublingual space below the geniohyoid muscle. Each is divided into two parts by the median septum.

is difficult. One cannot temporize in the management of these cases but must decide upon immediate surgical drainage if the patient is to be given his best chance of recovery. Infections involving these spaces usually have little tendency to localize as it is much easier for the infection to spread along the fascial planes. The infections usually are caused by streptococci or by a mixture of streptococci and staphylococci, with occasionally a few mouth organisms which produce gas and spread rapidly along the fascial planes. Contrary to the opinion of Alden[1] that most of these infections are caused by Vincent's organisms we have never yet been able to demonstrate these or-

ganisms in any of our cases of deep infection. He advocates the use of neoarsphenamine intravenously as the method of choice in the treatment of these cases, but we feel that this is not only worthless but subjects the patient to the toxic effects of a drug which in all probability actually lowers his resistance to the infection. Early incision and drainage is imperative, and this is best done through an incision made transversely across the neck between the symphysis of the mandible and hyoid bone. Through this incision the superficial and deep sublingual spaces can be easily explored by blunt dissection (Fig. 9). Any attempt at drainage which does not open into these spaces may result in failure. Rubber dam drains should be inserted and a continuous hot wet dressing applied. The maintenance of a normal fluid balance, general supportive treatment, and transfusions are of the utmost importance. A Mosher tube and a tracheotomy set should be kept in readiness at all times in anticipation of an acute respiratory obstruction. The infection from the deep and superficial sublingual spaces may pass to the lateral pharyngeal space and from there to the carotid space or to the pretracheal space and rapidly spread to the mediastinum with death finally ensuing from mediastinitis or general sepsis.

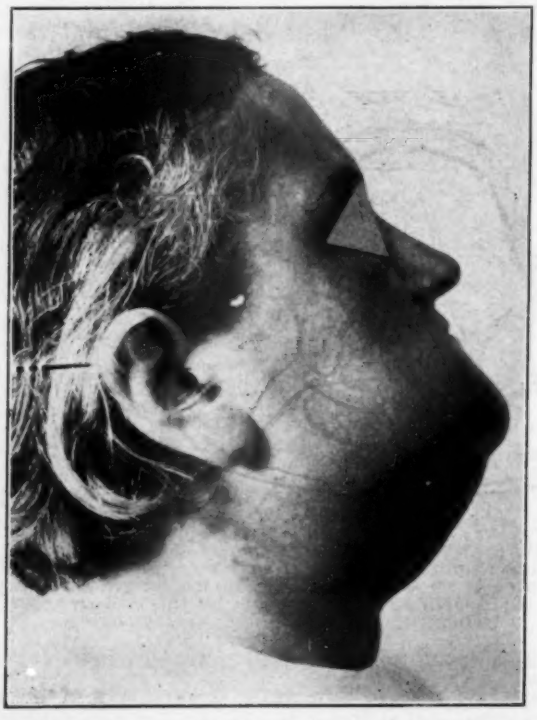

Fig. 8.—Acute infection of the superficial sublingual space. Most of the swelling is in the neck underneath the mandible. The pus was found between the mylohyoid and the geniohyoid muscles.

General Considerations.—It is very important to formulate and to follow out a certain definite plan of general treatment of cases with severe acute infections. The details of the plan may be altered to suit the individual case in hand, but in general, the following routine is carried out:

REST.—The patient is strictly confined to bed and kept as inactive as possible. Care is taken to make the patient comfortable by means of elevating the head of the bed and by the use of additional pillows. Visitors should be limited.

DIET.—A soft or liquid diet is usually necessary because of the patient's inability to chew or swallow without difficulty. This should be a diet high in calories and high in vitamins. Feeding by a nasal tube becomes necessary in some cases.

FLUIDS.—An attempt should be made to establish a normal fluid balance if the patient is at all dehydrated and to maintain this fluid balance throughout the course of the illness. If it is impossible for the patient to take adequate fluids by mouth, 5 per cent glucose should be administered intravenously. Under ordinary circumstances the average patient will lose about 2000 c.c. of fluid daily by evaporation from the skin and respiratory tract. In order for the kidneys to excrete the usual 35 gm. of waste products in a twenty-four-hour period with the least amount of effort, a minimum urinary output of 1500 c.c. is necessary.[8] This means that for maintenance of a normal fluid balance a total of 3500 c.c. of fluid per twenty-four-hour period must be given. If there is loss of fluid from draining wounds, blood loss, diarrhea, or vomiting, this must also be taken into consideration and allowance made to compensate for it.[5]

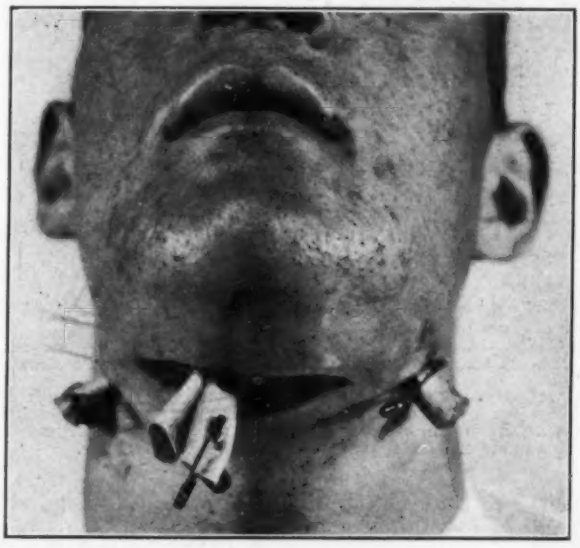

Fig. 9.—Incisions for drainage of neck infections should follow the normal folds of the skin in order to give the best ultimate cosmetic result. Both the superficial and the deep sublingual spaces were drained through this incision. The drains pass from the main incision through counter incisions at the angle of the mandible.

TRANSFUSIONS.—In cases of severe infection we feel that by giving repeated blood transfusions of 250 to 500 c.c. every other day the resistance of the patient is increased and convalescence is hastened by the additional antibodies given in the blood.

ELIMINATION.—If bowel movements are not regular, special consideration should be given to promote normal elimination.

SEDATION.—In patients who are not suffering from very severe pain it will be found that less potent sedatives will give relief. We, as physicians and dentists, have the drugs at our command that are capable of giving relief from pain, and the law has given us the right to administer these drugs as we see fit. In no case should we allow patients to suffer unnecessarily, but at all times should we make use of our prerogative to administer drugs. When narcotics are indicated, they should be given in the full therapeutic dosage. In many of these infections the pain is severe, and opiates are required for its relief.

Fig. 10.—A hot wet dressing to be effective must be large enough to hold considerable moisture. Dakin's tubing is incorporated in the dressing and saline solution is passed through it at frequent intervals to keep the dressing moist. Oiled silk incorporated near the outside of the dressing keeps the moisture from passing onto the bedclothing.

DRESSINGS.—We feel that continuous hot wet dressings should be used in cases of severe infection. Normal saline is preferable, but magnesium sulfate may be used. The use of this type of dressing is physiologically sound, as by the application of heat the blood supply to the affected part is stimulated. The increase in the blood volume flow to the area promotes the evacuation of toxic products from the region of infection and also increases the number of phagocytes and antibodies in the area. Except for its use in postoperative cases to prevent edema, ice has no place in the surgical armamentarium. A hot wet dressing in order to be effective must be made large enough to hold considerable heat and moisture. The dressing is made by the use of several gauze fluffs adjacent to the skin. These are covered by a large square abdominal pad. Dakin's

tubing is incorporated, and the dressing is then covered by a piece of oiled silk. This is then held in place by the use of two- or three-inch gauze bandage. Dressings should be neat and so constructed that they will not slip off, as there is nothing more disconcerting to a patient than an uncomfortable, slovenly looking, loose bandage. Saline solution or magnesium sulfate solution should be passed through the Dakin tubing at hourly intervals in order to insure adequate moisture. A heating pad or hot water bottle is kept over this dressing at all times (Fig. 10).

DRAINAGE.—We have had our best results with a tubular rubber dam drain material. Following external incision for the drainage of infections rubber dam has the advantage of not easily becoming crusted over. Gauze drains when crusted with blood and pus sometimes inhibit drainage. Iodoform gauze impregnated with vaseline is preferable for intraoral use.

X-RAY.—X-ray therapy should be routinely used in cases of deep infection of the type described in this paper. In many of the early cases the infection may be aborted by irradiation and in the late cases the resolution and localization of the infection will take place much more rapidly.

SULFANILAMIDE.—Since the advent of sulfanilamide the results obtained have been very encouraging in the treatment of acute infections about the face and jaws. By its use early I feel that we have aborted numerous infections that might have become widespread processes with a grave prognosis. The high incidence of *Streptococcus hemolyticus* in infections about the face makes it advisable to use the drug early even though the offending organism has not been determined. If the organism is found to be other than *Streptococcus hemolyticus,* no harm has been done. If *Streptococcus hemolyticus* is present, the infection may be aborted or brought in check without serious consequences. This drug is most effective when used early in the course of the disease. It serves the purpose of holding the infection in check until the natural defense forces of the host have been mobilized to the point where the infection can be overcome. Sulfanilamide is best given after the method of P. H. Long and E. A. Bliss[9] which is as follows:

Adults.—3 to 5 gm. (45 to 75 grains) initial oral dose, followed in six to eight hours by 1.0 to 1.5 gm. (15 to 22 grains), repeated every four hours.

Children.—50 to 90 pounds: 2 to 3 gm. initial dose followed as above by 0.6 to 1.0 gm.

Children.—25 to 50 pounds: 1.3 to 2.0 gm. initial dose followed as above by 0.3 to 0.6 gm.

Sodium bicarbonate should be given in equal amounts with each dose of sulfanilamide.

To get the maximum effect of the drug, a large initial dose should be given, and this should be followed by smaller amounts to maintain the optimum blood level. This dosage is maintained until definite improvement in the clinical course is noted, then decreased by one-third and again decreased by one-

third and continued at this level until the infection has subsided. The patient must be kept under close observation for the appearance of signs and symptoms of toxicity of the drug.

SUMMARY

1. A comprehensive knowledge of the anatomic spaces involved is imperative in the rational management of acute infections about the face and jaws. The various potential spaces of the face and the management of infection in these spaces are discussed.

2. Dental disease is the most prominent etiological factor in acute infections involving the space of the body of the mandible.

3. It is dangerous to extract teeth during the stage of acute infection. Conservative measures are recommended.

4. The masticator space is most often involved by extension of infection from the mandibular third molar area. Drainage should be delayed until the infection has localized.

5. A program of watchful waiting may result in failure in the management of acute septic parotitis.

6. Infection of the lateral pharyngeal space may result in septic thrombosis of the internal jugular vein, erosion of the carotid artery, or spread of infection to the fascial planes of the neck and mediastinum.

7. The sublingual spaces are most frequently involved by infection spreading from the space of the body of the mandible. Early incision and drainage are imperative.

8. General supportive measures are important in the management of acute infections of the face and jaws. These are considered briefly.

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A NOTE ON A CHROMOGENIC MICROORGANISM ISOLATED FROM AN ORANGE COLORED DEPOSIT ADHERING TO TEETH

HENRY A. BARTELS,* B.S., D.D.S., NEW YORK, N. Y.

A PATIENT, S. E., has been visiting my office semiannually for the past few years for oral examinations. She is about 35 years of age and particularly careful of her mouth hygiene. No visible calculus is observed about the necks of her teeth, and the gingiva has a healthy character in tone and appearance. However, on the labial surface of the mandibular central and lateral incisors in the cervical region there occurs a yellowish-orange deposit. This deposit may extend mesially and distally on the tooth, bordering the interproximal papillae. No other teeth in her mouth are involved.

The deposit in contrast to that of the green stain so often found on children's teeth is readily removed from the tooth surface with a scaler and brush. It appears somewhat granular and is orange yellow in color. Following its removal it again appears within a period of weeks to several months. When this condition was first noted I thought possibly some of the woman's lipstick had been in contact with the tooth surface. This idea was abandoned, however, when it was noted that the discoloration could not be removed with a pledget of cotton.

Under as sterile precautions as possible, some of the material was removed on a sterile scaler and inoculated on blood agar and Sabouraud's medium. After several days' incubation small, round, slightly raised colonies, some yellow, others orange in color, developed on the blood medium together with some colonies of the *Streptococcus viridans*. These colonies were fished and pure cultures obtained of organisms, one of which produced an orange pigment while the other grew with a yellow pigment. Both cultures were studied further.

Morphology.—Stained by Gram's method both microorganisms were found to be Gram negative pleomorphic bacilli which possessed neither capsules nor spores. Both bacilli were found to be motile.

Cultural Characteristics.—Growth developed readily on all simple media and also at room temperature (30° C.). No growth was obtained anaerobically.

Extract Agar.—The colonies appeared round, slightly raised, and uniformly granular. They became pigmented after several days.

Extract Broth.—A slight cloudy growth developed within twenty-four hours, which soon was followed by the formation of a pellicle. The pellicle was either orange or yellow depending on the microorganism inoculated.

Litmus Milk.—Slight alkalization of the milk occurred. No clotting was evident during a period of two weeks' incubation.

Gelatin.—Liquefaction of the gelatin occurred after a period of ten days.

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Potato Slant.—Within several days following inoculation either a yellow or an orange growth appeared.

Carbohydrates.—No fermentation was noted in Andrade's peptone water containing either glucose, lactose, maltose, saccharose, mannitol, xylose, or arabinose. The fermentation tests were observed for two weeks.

Indol.—No indol was formed.

It is evident that both the yellow and the orange colored bacilli possess similar morphologic and cultural characteristics, probably being strains of the same microorganism.

Animal Tests.—Injection of suspensions of the organisms into the peritoneal cavity of mice and guinea pigs caused no observable symptoms. The incisors of these animals were rubbed with loopfuls of the organisms, but no chromogenic deposit developed during a period of one month.

Identification.—Bergey's *Manual of Determinative Bacteriology* was consulted in an attempt to identify the organisms. These microorganisms belong apparently to the Genus Flavobacterium and closely approach, in characteristics, the *Flavobacterium lutescens.* This bacillus, however, is listed as nonmotile. Its habitat is water, and it is a saprophyte.

Evidently the patient has some local environmental factor which makes the mandibular anterior teeth a favorable site for the proliferation of these organisms. Such a deposit has not been observed on the teeth of her husband or daughter, although their diet is similar. The isolation of the yellow and orange colored colonies was successful at the first attempted cultivation. At a subsequent attempt only the orange pigmented microorganism was obtained.

The writer wishes to emphasize at this time that other microorganisms may also produce orange colored deposits. Just what factors favor their growth in some mouths and not in others is still unknown.

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HYDROTHERAPY

STERLING V. MEAD, WASHINGTON, D. C.

HYDROTHERAPY is the application of water in its various forms to the surface of the body to modify physiologic and pathologic processes.

Water acts as an ideal medium for the application of varying temperatures directly to the skin. The sensory nerve endings are affected by these variations of temperature, and result in certain reflex impulses which produce a phenomenon on the part of the body termed "reaction."

Water has the ability to hold certain salts and minerals in solution; therefore, it may also modify its effect upon the body. Water absorbs heat very rapidly and gives it up as quickly to bodies with which it is in contact, and it is considered an ideal medium for the administration of external heat.

On unbroken skin, or where the purpose of the application is to draw out the edema by osmotic action of the solution, magnesium sulphate in a saturated solution is indicated. The reduction of the edema inhibits bacterial action by depriving the bacteria of cultural media.

When there is an open wound and an astringent effect is also desired, aluminum acetate (Burow's solution) or aluminum subacetate may be used.

The compressing medium should have sufficient absorbent qualities and should be large enough to properly cover the area under treatment. Unless it is desired to have the medicament in the compressing solution absorbed, the skin should be covered with cold cream or vaseline.

The compress should be covered with oil silk, some nonabsorbent material, or with a towel, to prevent the moisture, which appears on the surface as condensation, from wetting the clothing, and also to keep the compress from changing temperature too rapidly.

HOT OR COLD APPLICATIONS

In reading the literature upon this subject one is impressed with the great variation of opinions. I believe, however, that the opinions of those of wide clinical experience differ more insofar as their mode of expression is concerned, and that fundamentally they agree upon most essential factors. In other words, when authorities differ they are often referring to different cases, but when a definite case is under discussion, it is usually found that they are in agreement.

Cold applications are used by many operators for the relief of pain, reduction or prevention of swelling, and inhibition of incubation of bacteria. In many instances cold does relieve pain. Cold is an aid in the prevention of swelling, and in some instances assists in the reduction of swelling. I am of the opinion that insofar as the bacterial regulating properties of cold applications are concerned, it is of little value. Cold undoubtedly causes contraction of the blood vessels and, in this way, lessens inflammatory reaction, tending to prevent suppuration, and also acts as an analgesic.

Attention has been called by many operators to the danger of reducing temperature too much, and there is danger of gangrene by refrigeration due to circulatory stasis. I believe that this danger is exaggerated.

Drury and Jones state: "Edema is two to four times greater at 42° C. than at 16° C. Therefore, it may be expected that applications of extreme heat may have a deleterious effect on local circulation and, therefore, on local processes."

There is a wide difference of opinion among authorities as to whether cold should be used continuously or intermittently. I believe that when cold is indicated, it should be used continuously, although this statement is misleading, as it is necessary to replace and refill the compressing agent or ice bag; in the average case, the demand is only for comparatively short periods.

The greatest good may be expected from cold applications when applied immediately after surgery where it is desired to prevent swelling, rather than in those cases where swelling has already occurred.

Cold, in some form, is useful in any injury likely to be followed by inflammatory processes. Federspiel aptly states: "The purpose of extraoral application of heat is to localize infection. Cold is used to limit the rapid destruction of tissue from acute inflammation. I see no reason why any hot or cold compresses should be applied after an operation. If the work is done properly and the tissues are not traumatized, and the patient is put to bed and given a sedative, there is no reason why hot or cold compresses should be used. Cold, of course, may be used to prevent the development of edema."

In surgery in the mouth, however, I find that at times there is necessarily some degree of undue trauma, as in the case of badly impacted mandibular third molars for patients with very dense bone structure, and in other similar types of cases. It may be due to retracting the tissues. These conditions are certainly analogous to the removal of tonsils, and surely the experience of the men in this field is that cold immediately after operation is helpful. I believe that cold is most useful in all of these cases when applied immediately and not one or two hours afterward. In these cases cold aids in preventing undue inflammatory reaction, pain and excessive bleeding.

In some of these cases, it is true that either hot or cold applications are efficacious. It has been my clinical experience, however, that heat in some of these cases does give very unfavorable results, causing excessive swelling, especially in those cases tending to bleed freely, and for this reason I do not believe in the extra-oral postoperative routine use of heat for these particular cases.

Some writers state that cold should be used only in inflammatory conditions and not where there is infection or where the condition is acute. These statements are also very misleading, as infection is no doubt associated with all wounds about the mouth, and there are various degrees of acute conditions. Now, I believe that there are certain infectious processes and certain stages of acute infection where cold will be of no advantage, and even a disadvantage; but, on the other hand, experience has shown me the advantage in some of these cases of using cold. After all, the use of hot and cold

applications cannot be exactly standardized, but must be timed as the case dictates, with a clear understanding of what is expected of it at the time.

My statement that cold applications can be used to advantage in the beginning stages of acute infection to prevent swelling, has been criticised by some writers. I refer in these cases to the stage of acute infection where suppuration has not commenced.

I have also been criticised for the statement "When swelling occurs in the presence of cold applications or continues more than twelve hours, alternating hot and cold applications may be used. Then, if the swelling continues, continuous hot applications should be used." I should, of course, qualify this statement by saying in some particular cases this method is advantageous; for, as a general rule, if the operator of experience does not know whether to use hot or cold, it is well not to use either one. As an illustration of the advantage of this method, I can cite my experience with the treatment of hematoma following injection of a local anesthetic. In these cases I immediately make an incision into the area traveled by the needle and spread the incision with a small hemostat. By applying alternating hot cloths and cold cloths, it has been my clinical experience that the swelling subsides more quickly, and with less discoloration than by other treatment. This has practically eliminated the danger of suppuration which is always a potential sequela which I formerly experienced in a small percentage of these cases. My impression is that this reduction of swelling is accomplished through physiologic reaction rather than bacteriologic means.

When it is suggested for a particular case that cold packs be used extraorally and hot solutions used intraorally, the two procedures often sound contradictory to some people. This is not necessarily so, because in some of these cases, the hot solutions are used in the mouth with the idea of hastening the inflammation process and drainage, with the use of cold externally in an endeavor to retard extraoral inflammation, swelling, and extension to surrounding tissues.

One is sometimes confronted with an emergency request, as in the case in which one is called at night by a patient in the country, where a dentist or physician is not immediately available, who wants to know whether to use heat or cold as an emergency treatment in order to tide him through the night. In a case of this kind where it is difficult to determine from a description of the case, without observing it clinically, just what is best to do, it is much safer to use cold as a temporary measure, because heat in a small percentage of these cases may cause undue swelling and spreading of inflammation and infection. This is, no doubt, what the physician or surgeon would do in a suspected case of appendicitis until he was able to see the patient and get a differential blood count, etc.

After the occurrence of exudation, cold hinders the evolution of the process and prevents absorption.

Moose states: "The activity of the leucocytes in phagocytosis is modified by temperature. Phagocytosis has been described as being maximal at the

normal temperature level of the individual, but, with fever, the optimal temperature is found to be the febrile level. Phagocytosis is impeded by local cooling.

"Metabolism is affected by temperature changes, cold having a deleterious effect, as can be observed readily by cyanosis on exposure to low temperature. An impediment to metabolism often affects tissue change, and muscle activity is slowed down by exposure to cold. The warming up of athletes is evidence of this fact.

"When using heat in inflammatory conditions, we must remember that vasodilatation increases fluid exudation, so that, under certain circumstances, heat may appear to be contraindicated, particularly when exudation will interfere with blood flow. If the rate of blood flow is the most important factor, moderate cold with vasoconstriction and a decrease in capillary pressure might produce more rapid blood flow than vasodilatation with fluid exudation and consequent venous congestion. Thus, the beneficial use of ice packs might be explained, and certain cases might be selected on which to use cold. In any case except when the above mentioned conditions are increased, the use of cold is contraindicated, since the physiologic processes of immunity and phagocytosis are slowed. Even when edema is very evident, cold may not be beneficial. Filhene, 1894, during his work on erysipelas, found cold applications of doubtful value; while the mortality rate was lowered and more rapid convalescence obtained with warm applications even though the local lesions were more acute and angry in appearance."

Gardner states: "From a strictly bacteriologic standpoint, the variance of temperature created by hot or cold surface application is of little consequence in inhibiting bacterial growth. If swelling and infection have become more extensive, heat, by means of hot compresses, is undoubtedly the choice. This is applied for the purpose of bringing about active hyperemia, and thereby increasing the number of leucocytes, hastening the formation of a leucocytic membrane and localizing the infection."

Blair states that: "One of the oldest treatments for localized infection is heat: In general it seems to promote comfort, allay pain, and promote the circulation. In septic infection, it probably predisposes to suppuration; but in the presence of a septic infection of a certain virulency, local suppuration cannot be regarded as an evil. According to our present ideas, it is rather difficult to explain the good that undoubtedly results in many instances from the application of cold to an inflamed part. It cannot be the result of direct action of the cold on the infecting organisms, which are generally too deep seated to be influenced by a direct cooling effect. Cold undoubtedly causes contraction of the blood vessels and lessening of the inflammatory reaction, and tends to prevent suppuration and allay pain. There are certain animal experiments that demonstrate the fact that reducing the temperature of the tissues reduces their resistance. We might conclude that, in all instances, the inflammatory reaction is excessive, and that cold is beneficial in regulating it."

Moose quoting McLean states: "McLean believes that all types of edema or inflammatory exudate of the maxilla or the mandible should be handled as potential cases of deep cellular infections. Scar formation on the face is

not desirable but more acceptable than bone destruction and general sepsis. In the treatment of osteomyelitis, he believes that heat is indispensable, and he employs it to the exclusion of everything else. For inflammations of short duration, i.e., from one to three days, when there is but slight extravasation of fluid and a great deal of swelling, and the fluid is apparently not deeply seated, he finds cold applications to be helpful, while using hot intraoral irrigation."

Berger states: "The processes of inflammation are in a large measure protective and defensive. Heat is more frequently indicated when there is stagnation and probably a deep-seated suppurative nidus. After free drainage is established, heat should be discontinued and moist, cold dressings will have a more beneficial influence."

Johnson states: "I am in agreement with the usual theory that the judicious use of cold is valuable in reducing hyperemia by causing contraction of the arterioles. . . . I believe that if used at all, it should be used early for the prevention of edema resulting from trauma or confusion in operative procedure in relatively noninfective cases. I believe that it should not be used if rampant infection or much congestion is already present, as cold depresses the vitality of the part and may do more harm than good. I believe that the judicious application of heat activates the vitality of the part by producing an active hyperemia, causing an increased vascular supply. The part is bathed in fresher, more oxygenated blood, which may reasonably be assumed to contain leucocytes, opsonins, antitoxins and other antibodies through which the bacteria and their toxins may be neutralized. Clinical experience also leads me to believe that heat relaxes the tissues and vessels, often reducing tension and pain. When applied to spastic muscles or to hard, infiltrated swelling, it assists in the restoration of healthy circulation, and softens and relaxes the tissues."

HEAT

Heat is one of the local effects which will cause tissue relaxation. The most marked result of the application of surface heat is that of analgesia, especially in local infections. Pain due to muscle spasm or cramp is in many cases promptly relieved by heat, as spastic muscles may often be directly relaxed when heated. Vasodilatation also occurs in varying amounts, affecting primarily the capillaries. The capillary wall is thinned, and the intercellular spaces are increased, which will permit the extravasation of a greater amount of blood serum into the tissues. The sensory nerve endings, which bring about circulatory changes in the deeper organs through reflex action, are directly affected.

Heat is not often used in the first stage of inflammation, because the amount necessary to contract the vessels is too great for comfort. In the later stages, heat which is comfortable to the patient relaxes the tissues, lessens tensions, relieves pain, assists absorption, and, in the presence of bacteria, hastens suppuration. In inflammations below the surface it acts as a counterirritant by diverting blood from the affected part.

Heat may be applied in the form of gauze or cloths immersed in hot water, hot water bag, electric pad, heat lamps, irrigants, and poultices.

The solutions most often used extraorally in the compressing vehicles are magnesium sulphate, aluminum acetate (Burow's solution), aluminum subacetate, and boric acid. A good solution for use intraorally is normal salt and soda solution or chlorazene.

COLD

Cold may be applied by the use of pieces of gauze or a cloth applied to the area after being immersed in ice water; by the use of ice chips or cracked ice in a rubber bag, ice collar, or by the use of special rubber bags especially made for freezing in an electric refrigerator.

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CELLULITIS

Case Report*

Robert C. Ingram, D.D.S., Laurel, Md.

March 12, 1939—The patient, a mongoloid, mentally deficient child about 8 years of age, had been in quite satisfactory health. He returned to school about a month ago and had had no complaints, except an occasional slight cold. Last week he was in bed about two days with a temperature, and some cough, but he recovered rapidly. Yesterday evening he was noted to be drowsy, and a temperature of 102° F. was recorded. He was put to bed, and phenacetin was given. This afternoon his temperature was still 102° F., respiratory rate was not increased, and chest examination was negative. There was a large *fever blister on the lower lip, surrounded by considerable swelling and induration.*

March 13, 1939—The swelling of the lower lip became much more marked, involving the whole lip, with excoriation of the mucous membrane, and many tiny pustules being present. The lip was treated by hot packs. The patient's temperature was 102° F., chest examination negative.

March 14 ,1939—The swelling of the lower lip was about the same. There was now, however, redness and induration of the entire chin, a definite erysipeloid appearance. A smear was taken from one small pustule; only a few organisms were found in the smear, but these were mainly diplococci, which in some areas arranged themselves in a line, as to resemble streptococci. The smear was stained with methylene blue, so whether the organisms were Gram positive or Gram negative was not determined. Chest examination showed definite dullness, with increased fremitus, and bronchial breathing, on the left side, more marked in the upper lobe. Sulfanilamide was ordered, 15 grains at 4:00 P.M., 8:00 P.M., 12:00 midnight, and 8:00 A.M.

March 15, 1939—The patient's condition was about the same. The entire mucous membrane surface of the lip was honeycombed with areas which were discharging pus. Extension of the infection seemed to have stopped, but the entire lip and chin had been involved, and pus formation was occurring. Sulfanilamide, 15 gr. at 2:00 P.M. and 8:00 P.M. was given. The left upper lobe of the lungs still showed signs of pneumonia, temperature ranging about 102° F.

March 16, 1939—There was quite profuse discharge of pus from multiple sinuses in the lip; no definite pointing on the chin was observed. The chest condition was about the same. The patient was able to take nourishment. Another smear from the lip was taken, and organisms were found only with considerable difficulty. It is possible that they had been killed by the sulfanilamide, and so were disintegrated.

March 17, 1939—The patient died March 17, 1939, at 10:30 P.M. The cause of death was cellulitis of the face and pneumonia.

*From the District Training School, Laurel, Md.

Case Reports

This case presented by Dr. W H. Hyde is a very unusual one. It presents a peripheral tumor of the fibroma type attached to the mandible. In addition, the roentgen film shows a change in the adjacent bone which generally is consistent with a bone tumor described as osteofibroma.

Case reports for this department should be sent to Dr. Kurt H. Thoma, 53 Bay State Road, Boston, Mass.

CASE REPORT NO. 28

OSTEOFIBROMA

WILLIAM H. HYDE, D.D.S., F.I.C.A., BROOKLYN, N. Y.

Chief Complaint.—Patient, M. G., female, born in the United States, aged 18 years, complained of a growth on the right gingiva of the mandible which had been present for approximately two years.

History.—The swelling on the gingiva had been present approximately two or three years and had slowly increased in size until it was about the size of a walnut. It had a pedicle and a broad base, and was normal in color and texture. There was no pain except for the discomfort and interference with her speech and eating. There was no adenopathy present; her lips and cheek were normal; her mouth was in a fair condition. A differential count revealed red blood cells

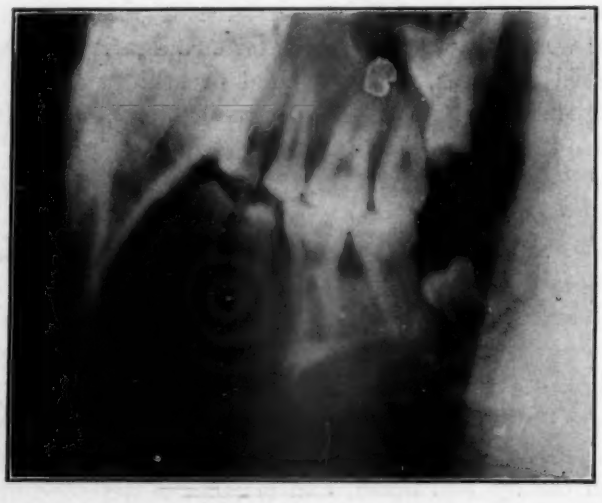


Fig. 1.

4,500,000, white blood cells 7,400, polys 81 per cent. Urine examination was negative for sugar and albumin, acid in reaction, all other features being negative. The smear showed only normal bacteria found in the mouth. Preoperative diagnosis: fibroma.

Roentgen Examination.—The mandible showed a definite change in the architecture of the spongiosa in the area from the first premolar to the third molar. The area was dense because the bone trabeculae were very fine and condensed (Fig. 1). This was consistent with a diagnosis of osteofibroma.

Treatment.—Under local anesthesia the mass was excised. It was found adherent to the underlying gingivoperiosteum. At no time during the operation was there any impression of cutting through either cartilaginous or bony tissue. The excised mass appeared vascular, but there was no undue hemorrhage from the incised surface.

Pathologic Examination.—Gross: The specimen was a nodular portion of firm gray and pink tissue, 2 × 5 × 1 cm., said to have been removed from the jaw. Part of the external surface was smooth and glistening; the remainder was rough. The cut surfaces resisted cutting and were pink streaked with gray.

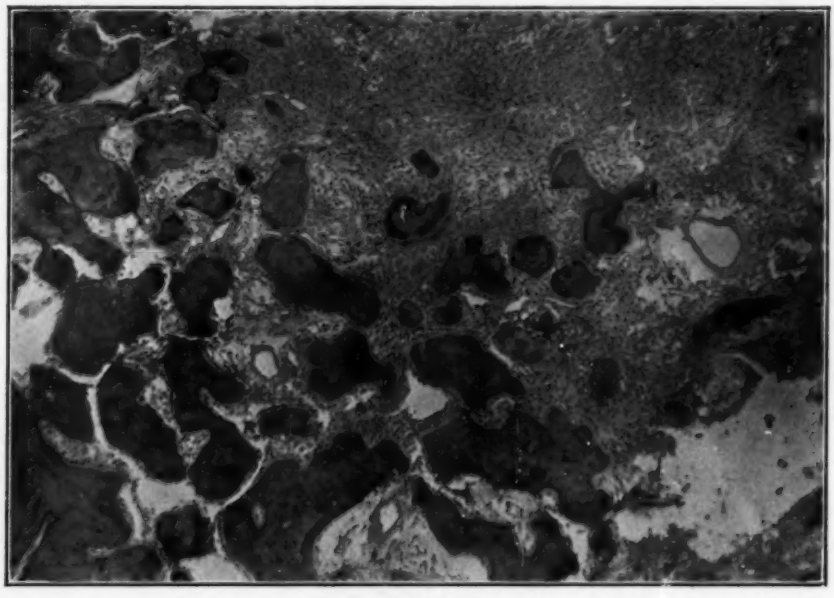


Fig. 2.

Microscopic: Parts of the surfaces of the different fragments were covered by stratified squamous epithelium of varying thickness. Immediately below this covering epithelium the connective tissue was loose, vascular, and in places infiltrated with small round cells, large mononuclears, and some polymorphonuclear leucocytes. In the deeper portions in places were seen broad and delicate bony trabeculae with well-formed Haversian systems. The edges in some were ragged and stained poorly. In other areas there were scattered deposits of calcium. The marrow spaces contained numerous fibroblasts and in places were filled with dense fibrous connective tissue. Here there also was evidence of round-cell infiltration.

Diagnosis.—Osseous and fibrous tissue from jaw with chronic inflammatory reaction.

The slide was reviewed in the Laboratory of Oral Pathology, Harvard University, by Dr. Thoma, who thought that the appearance of the tissue (Fig. 2) was consistent with that of ossifying fibroma.

Department of Orthodontic Abstracts and Reviews

Edited by

Dr. J. A. Salzmann, New York City

All communications concerning further information about abstracted material and the acceptance of articles or books for consideration in this department should be addressed to Dr. J. A. Salzmann, 654 Madison Avenue, New York City.

Facial Disfigurement and Personality. By W. Y. Baker, M.D., and L. H. Smith, *J. A. M. A.* **112**: 301-304 (Jan. 28), 1939.

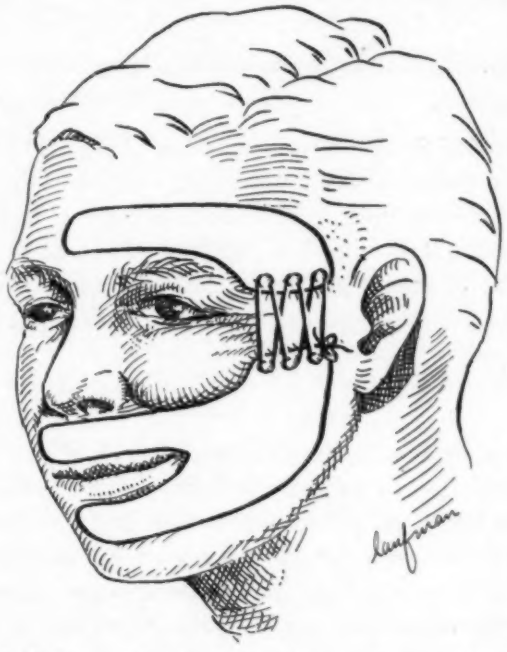
Plastic surgeons have seen patients whom they have benefited both in body and in mind but who have remained unsatisfied and have shown that they are sick and maladjusted persons. A study was undertaken at the Graduate Hospital of the University of Pennsylvania of facial disfigurement, under the direction of Dr. Robert H. Ivy and his associates. There has been a total of 312 such patients. Patients with disfigurements of recent traumatic origin were not included. From two to five years is necessary for the personality to accept and incorporate the new factor.

A consideration of the personality and emotional status of the patient seeking a facial operation is recommended in an attempt to diminish the number of emotional defects which hound the surgeon after good technical results have been obtained. This can be achieved by a better understanding of mental and personal mechanisms on the part of the plastic surgeon and can be further facilitated by a closer association of the plastic surgeon and the psychiatrist. An estimation of the personality by one trained in that field as well as in the field of general medicine is recommended at least until the surgeon has acquired an insight and a workable concept of what personality factors influence the total result from the patient's standpoint.

In this connection, orthodontists can think back on the numerous occasions when patients with comparatively minor dental malformations have presented themselves for treatment. The orthodontist later finds, much to his regret, that he has let himself in for endless argument and vexation which eventually lead to a strained patient-doctor relationship, if not actual suit. Orthodontists would do well to give heed to the mental and emotional state of the patient before undertaking treatment of apparently minor cases, especially in adults. Furthermore, the orthodontist should make it clear to the patient that, while the dental malocclusion might be corrected, its effect on facial appearance cannot be foretold and furthermore that the malformation might recur.

The Preservation of Muscle Function in Bell's Palsy. By P. Lewin, M.D., Chicago, *J. A. M. A.* **112**: 2273, June 3, 1939.

Lewin describes a splint made of adhesive plaster, designed at the Michael Reese Hospital, Chicago, which he used for supporting the facial muscles in a case of Bell's palsy. A strip of tape was placed across the forehead extending to the temple on the affected side. A second strip was slit at one end and placed over the skin above the upper lip and across the chin. The strip was drawn across the affected side of the face. Hooks such as are used by gynecologists were attached at the adjacent ends of each of the adhesive strips near the temple, and the two laced together, producing an overcorrection of the muscular deformity. After 10 days of splinting the patient was given a series of galvanic treatments. Complete recovery followed.



Splint for supporting the facial muscles in Bell's Palsy. (From J. A. M. A. 112: 2273, 1939.)

The Role of Allergy in the Etiology of Orthodontic Deformity. By T. W. Todd, M. B. Cohen, and B. H. Broadbent, *The J. of Allergy,* March, 1939.

In a study of sixty children under treatment for allergy the authors noticed orthodontic deformity in 45 or 75 per cent. The causes of these malformations are the result of four processes:

1. The ultimate size and general form of the jaws are determined by a hereditary pattern. Environmental influences determine the extent to which the innate growth limits are attained.

2. The growth in stature of the infant, in the first year of life, is the same as that during four and a half years of later childhood. In infancy growth is most rapid in the head. At birth the brain is but one-fifth adult size; by the end of the year it is two-thirds as large as the adult brain. More than

one-half of the postnatal growth of the brain occurs in the first year. The face grows more slowly, but since the face is an appendage of the cranium, it cannot fail to follow the cranium in an extraordinarily rapid growth.

3. All rapidly growing structures are vulnerable, and, therefore, the face is in greatest danger of mutilation during the first year. The cause of this defective and deformed growth is malnutrition in its broadest sense.

4. The face grows in all three directions simultaneously. Disturbances of growth affect all three dimensions, although only one or two may show major effects. In those infants who suffer from nasal disorders during the first half year of life the face growth will be markedly retarded in all three dimensions. Those who suffer for the first time during the second year have less marked deformities, especially the lateral expansion of the hard palate. Those who suffer at five or six may show only a slight diminution in either forward or downward growth.

Traumatic Occlusion. By John Oppie McCall, New York City, *J. A. D. A.* **26**: 519-526, April, 1939.

Traumatic occlusion is the most intangible thing in the mouth. The pressure tolerated in one mouth without harm may cause serious harm in the next one. The existence of traumatic occlusion is readily visualized when gross malocclusion is present, but it is less easy to conceive of its existence when the occlusal relations of maxillary to mandibular teeth seem to be normal or nearly so. Yet some of the most serious manifestations of this condition are to be found in the latter type of case. Occlusion is traumatic when the supporting tissues of the teeth involved undergo pathologic change as a result of occlusal stress.

Any malposition may cause traumatic occlusion if it is of such nature that either in centric occlusion or in the various contacts incident to mastication, there is an uneven distribution of stress throughout the arch. Prominent examples are excessive overbite of the anterior teeth and abnormal mesiodistal relations of the maxillary and mandibular premolars. Less common, but still more injurious, is the occasional malposition in which a maxillary premolar, for instance, closes entirely to the buccal aspect of the mandibular tooth. Compensating wear is normally accomplished through the vigorous mastication of coarse, fibrous foods. Since civilization has largely eliminated such foods from the dietary, but little natural wear is seen in the teeth of modern people.

We must resort, in less severe malposition and malformation, to grinding at the hands of the dentist. Orthodontics must come to the rescue in many cases. There is the alternative of providing the patient with a chewing gum charged with abrasive powder, to be used under careful supervision. Grinding is the method most commonly used in the correction of traumatic occlusion. In many cases grinding can give only partial relief, particularly when there is an excessive overbite of the anterior teeth. Here, orthodontic treatment is indicated, and this may be done not only for the young, but even for the middle-aged. Opening of the bite is not successful in these cases except when combined with orthodontic treatment and then is usually not needed.

In the case of incipient periodontal disease, if grinding is to be done, it is usually best to start with the anterior teeth and bring them into a state of balance in both centric and protrusive occlusion. Establishment of balance in centric position is determined by placing the tips of the fingers very lightly on the maxillary incisors, while the patient closes the teeth rather smartly. There should be no displacement nor even a marked jar as the teeth make contact. It is usually well even to create a slight space so that a sheet of tissue paper can be drawn from between these teeth without tearing. This space will soon close up by elongation, and such growth will cease when the teeth just begin to make contact once more, and there will thus be no re-establishment of the traumatic occlusion.

Book Reviews

The Immortal Tooth. By Edward Samson, London, Eng., John Lane the Bodley Head, 1939.

Edward Samson is no stranger to most well-informed American dentists. He is a British dental writer of note on the scientific as well as the historical and whimsical aspects of dentistry. The present volume of *anecdota dentorum* could well serve as reception room literature with the double advantage that it would not soon be out of date and would tend to arouse among patients interest in teeth not necessarily associated with bodily pain. Dentists themselves will read the book with great interest. However, there is a practical basis underlying the writing of this book. Quite unaware, before he realizes it, the reader is knee-deep in sound educational dental health advice. Authorities, including most of the American writers past and present, are quoted on the relationship of the teeth to general health.

One would like to quote a few samples of the dozens of interesting tales about teeth. However, Samson has taken good care to pick stories of uniformly high interest. We advise readers to buy the book and read it from cover to cover.

Cheilosis Due to Ariboflavinosis. By W. H. Sebrell and R. E. Butler, Riboflavin Deficiency in Man, *Pub. Health Rep.* **53**: 2282 (Dec. 30), 1938.

Each day, as new discoveries are made, the responsibilities of the dentist increase. It is now found that probably the earliest sign of riboflavin deficiency, as with deficiencies of most of the other vitamin and mineral constituents of the diet, appear first in the purview of the dentist. Sebrell and Butler, by the use of a riboflavin-deficient diet, produced a cheilosis which was initiated with a pallor of the mucosa of the lips at the angles of the mouth. This did not involve the mucous membrane of the buccal parietes. Whether or not this is the same condition as has been previously described as *la perlèche,* it must be borne in mind that at least a susceptibility to the cracking and fissuring of the angles of the mouth may be produced by a deficiency of this fraction of vitamin G.

It was found that the administration of 1 or 2 mg. of synthetic crystalline riboflavin altered after three to ten days to 0.25 mg. per kilogram of body weight, eliminated the lesions in five to forty-seven days. Also, it is inter-

esting to note that the aberration can be controlled by the administration of riboflavin and yet is not improved by the use of nicotinic acid (the pellagra-preventing fraction of vitamin G).

Sebrell and Butler believe the condition which they term "ariboflavinosis" should be added to the list of vitamin deficiency diseases with the lesions at the angles of the mouth as the chief clinical symptom. This presents to the dentist a new approach to the handling of lesions at the angles of the mouth which previously had been attributed almost entirely to streptococcic or yeast infection.

Samuel Charles Miller.

Hydrogen Peroxide and Sodium Perborate: Their Comparative Oral Irritant Action. By Samuel Charles Miller, Sidney Sorrin, William M. Greenhut, and Rudolph H. Pelzer, *Bul. N. Y. State Dent. Soc.*, Vol. 6, No. 2, 1939.

The authors recommend the use of the scientific term "Lingua Filaceosa Chromatica" instead of the terms black tongue, hairy tongue, and black hairy tongue. By practical experiments performed on 181 dental students it was found that 21.1 per cent of those who used sodium perborate in solution as a mouthwash developed the "Lingua Filaceosa Chromatica." Only 6.8 per cent of those using dilutions of hydrogen peroxide developed this tongue abnormality. Sodium perborate is extremely alkaline (pH 9.3 to 9.9) and is affected little by the addition of sodium bicarbonate or by mixing with saliva in the mouth. Hydrogen peroxide, on the other hand, is slightly acid (pH 6.0 to 6.3) and is quickly buffered by mixing with saliva in vivo.

The possible production of oral pathosis by the use of these substances indicates that they should be used only under supervision as specific therapeutic agents. Patients should be given specific instructions as to the concentration to be used, the frequency and method of use, and the rinsing of the mouth with water after the solutions have contacted the oral tissues. Systemic effects of excessive use of sodium perborate as a mouthwash are possible.

D. Tanchester.

The Surgical Risk in Patients With Coronary Disease. By H. J. Brumm, M.D., and F. A. Willins, M.D. *J. A. M. A.* **112**: 2377-38, 1939.

To a considerable degree, the results of surgical intervention are dependent on the expertness and finesse exercised in the management of the patient by all concerned. This includes not only the surgeon but likewise the anesthetist and clinician. The shorter the time of operation and anesthesia, the less marked is the added load imposed on the cardiovascular system. Likewise, gentleness in the manipulation of organs and tissues lessens the dangers of surgical shock, which in itself may tilt the balance away from recovery in these patients. Of paramount importance is the surgeon's determination to limit the operation to the procedure planned in advance and to avoid undertaking additional operative steps that might be indicated in the patient without heart disease.

Two hundred and fifty-seven patients with severe coronary disease undergoing necessary surgical operation form the basis for this study. The average age of the patients was 60.3 years. The anginal syndrome had existed for an average of 3.1 years. Thirty-two patients had healed cardiac infarcts at the time of operation. Well-marked hypertension occurred in 100 cases. Eleven patients (4.3 per cent) died from cardiac causes. Death resulted from coronary thrombosis in seven, from congestive heart failure in two and abruptly without apparent thrombotic occlusion in two.

The cardiac mortality in the group of cases is remarkably low and should encourage the sufferers from coronary disease when surgical intervention is necessary. However, it must not instill false optimism into the clinician or the surgeon, for they must realize that this accomplishment is not of casual origin but one resulting from the coordination of careful preoperative study and judicious selection, expert administration of anesthetic agents and skillful surgical technic and judgment. Operation must be confined to those cases presenting unmistakable indications and the procedure limited to the primary condition. Surgical procedures that are not urgent have no place in the cases under discussion.

Editorial

Nutrition and Bone Form

NO ONE, as yet, seems to know to what degree not only the progress but also the entire physical destiny of a race depends upon its diet and, incidentally, its nutrition. Notwithstanding, pieces are slowly, step by step, being put together which sharpen out the picture that must sooner or later add greatly to the whole knowledge of nutrition, its resultant growth and skeletal formation.

There seems to be little doubt that body ailments peculiar to the present civilization have a definite "tie-in" with the modern diet, to which civilized man has adapted himself over a period of many years.

A doctor, who is practicing near the Mediterranean Sea in approximately the desert region, is trying to find out, for instance, why people who live on the desert nearby are so rarely afflicted with appendicitis. Another is making a serious effort to ascertain why epidemics of gall bladder trouble occur in certain districts, and why they are so rare in others.

Weston A. Price, D.D.S., has spent many years visiting in faraway places of the world and in his current book, *Nutrition and Physical Degeneration,* he reports findings that are revealing not only to the layman but also to the professions. He says, for instance, that wherever the natives subsist on their own original and extraordinary diet, good teeth and a hardy people were found. In the main, when the aborigines adopted the white man's diet, they found the white man's troubles, particularly manifested in dental caries and deformities of the alveolar areas.

Doctor Waugh reports much the same situation, after having spent much time with the Eskimos of the Far North. Doctor Price has further reported that many primitive peoples have learned the specific value of certain foods when fed to prospective mothers and to children, in order to avoid the deficiency diseases, such as scurvy and rickets, that are common in modern civilizations.

A number of incidents are being reported wherein it is pointed out that primitive people, when removed only one generation and forced to live on the civilized man's diet, are afflicted with a wide array of both dental decay and maxillary facial deformities.

Inferentially, at least, this would tend to back up the statement which has been made informally by a great orthodontic mentor. Dr. John Mershon is the author of the statement that orthodontic treatment must not exceed the growth velocity of nutrition and bone, otherwise relapse often occurs.

It is not fantastic to assume that orthodontists will someday become so nutrition-minded that diet will become at least as important in treatment of

malocclusion in children as the relative position of the mesiobuccal cusp of the maxillary first molar or the status quo of adenoids and tonsils has been in the past.

More than one orthodontist who has spend many years correcting malocclusion is left in a quandry of thought and speculation after reading the evidence presented on the subject of nutrition and its effect on facial form in savage races.

Many orthodontists would, no doubt, answer the evidence presented by saying that nutrition may have been no more responsible for a change in facial bones in one generation than environment, that is to say, hard physical labor and constant physical effort contribute to large strong bones and vice versa. The evidence presented makes interesting reading for the orthodontist, on account of its array of evidence presented on the subject of osseous changes in the face and head.

H. C. P.

News and Notes

Mexican Association of Orthodontia

The Mexican Association of Orthodontia held a meeting in the city of Mexico, with the assistance of medical surgeons and dental surgeons, national and foreign, from May 22 to 26, at the National Faculty of Odontology, Lic Verdad St. and Guatemala St.

PROGRAM

Presidency: Dr. Gus Baz, rector of National University of Mexico.
Greetings: Dr. Fransisco Calderon Caso, president of the Mexican Association of Orthodontics.
The Endocrine Factor in Relation to Medicine and Dental Surgery: Dr. Ralph Raynolds, M.D., San Francisco, Calif.
A New Articulator: Dr. Carlos Zepeda, D.D.S., San Salvador, Central America.
Presidency: Dr. Don J. Aubertine, member of the Public Health Committee of A.D.A.
Mobilization of the Natural Teeth: Evolution of the Appliances: Dr. Fransisco Calderon Caso, member of National Academy of Medicine and Dental Surgery.
Practical Methods for the Construction of Crowns and Bridges: Dr. Harold Carson, D.D.S., Eureka, Calif.
Presidency: Dr. Antonio Guerrero, dean of the National School of Orthodontia.
Problems of Oral Pathology: Dr. J. Aubertine, D.D.S., San Francisco, Calif.
The Technique of Atkinson and the Application in Orthodontics: Dr. S. Fastlight, D.D.S., Mexico.
Presidency: Dr. Jose Aguilar Alvarez, director of Medical School.
Hyperthyroidism in Relations to the Diseases of the Teeth: Dr. Fransisco de P. Miranda, Med. Surgeon, Mex. F.A.C.P., professor of Clinics, member of National Academy of Medicine.
Crown and Bridge: Dr. Harold Carson, D.D.S., Eureka, Calif.
Presidency: Dr. D. Leonides Andrew Almazan, head of Public Health Dept.
The Medicaments, Their Studies and Their Application in Dental Surgery: Dr. Edward Mimmack, D.D.S., Buffalo, N. Y.
Fracture of the Maxilla; Prosthetic Treatment; Adult Prothesis: Dr. Alejandro Velasco Zimbron, prof. Buccal Surgery, Mexico.
Closing Speech: Dr. Rafael Ferriz, secretary of the Mexican Association of Orthodontia.

Mexican Association of Orthodontia, 1939

Honorary President: Dr. Spencer R. Atkinson
President: Dr. Fransisco Calderon Caso
Secretary: Dr. Rafael Ferriz
Treasurer: Dr. Joaquin A. Casasus

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Dr. Samuel Fastlight
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Dr. Ralph Horne
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Dr. Carlos M. Paz
Dr. Jose M. Rojo

INVITED MEMBERS OF HONOR

Dr. Leonides Andrew Almazan, Head of Dept. of Public Health.
Dr. Gustavo Baz, Director of Medical School.
Dr. Antonio Guerrero, Director of National School of Orthodontia.
Dr. J. Aubertine, member of Health Committee of American Dental Association.

Great Lakes Association of Orthodontists

The thirteenth annual meeting of the Great Lakes Association of Orthodontists will be held at the Dearborn Inn, Dearborn, Mich., on Nov. 6 and 7, 1939.

Biological Photographic Association

The ninth annual convention of the Biological Photographic Association will be held Sept. 14 to 16, at the Mellon Institute for Industrial Research, Pittsburgh, Pa. The program will be of interest to scientific photographers, scientists who use photography as an aid in their work, teachers in the biological fields, technical experts, and serious amateurs. It will include discussions of motion picture and still photography, photomicrography, color and monochrome films, processing, etc., all in the field of scientific illustrating. Modern equipment will be shown in the technical exhibit; the Print Salon will display the work of many of the leading biologic photographers here and abroad.

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Secretary-Treasurer, Charles R. Wood ..... Knoxville, Tenn.

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James D. McCoy ..... Los Angeles, Calif.

Foreign Societies†

British Society for the Study of Orthodontics

President, S. A. Riddett
Secretary, R. Cutler
Treasurer, H. R. Evans

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*The Journal will make changes or additions to the above list when notified by the secretary-treasurer of the various societies. In the event societies desire more complete publication of the names of officers, this will be done upon receipt of the names from the secretary-treasurer.

†The Journal will publish the names of the president and secretary-treasurer of foreign orthodontic societies if the information is sent direct to the editor, 8022 Forsythe, St. Louis, Mo., U. S. A.